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The next Annual Meeting of the Massachusetts Medical Society will be held at Plymouth, June 17 and 18, 1930.

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The Massachusetts Medical Society

SECTION OF MEDICINE

TUESDAY MORNING, JUNE 11, 1929

THE Section of Medicine of the Massachusetts Medical Society, held in the Ballroom of the Copley-Plaza Hotel, Boston, Massachusetts, convened at nine-twenty o'clock, Dr. Lester C. Miller, of Worcester, Chairman, presiding.

CHAIRMAN MILLER: *Gentlemen of the Medical Section*—We are rather late in starting, partly due to the fact that our apparatus for showing pictures was not ready.

We are obliged to change the program a little bit from the printed order, because Dr. Cole, who is showing some pictures here and talking on the X-ray problem, is obliged to go from here to the Section of Physiotherapy. Consequently, we have to take him on earlier in order that he may finish his subject in there. I will say to the disadvantage of this Section, if you want to follow his subject through after he has given what he has to say here, it will be necessary for you to go into the Physiotherapy Section, however much we dislike to lose you.

The first speaker this morning is so well known to this Society that it is hardly necessary to introduce him. He is perhaps better known for his clinical investigations along the line of gastro-intestinal diseases, particularly stomach and cancer, and as I remember some very interesting studies in occult blood. It gives me pleasure to introduce Dr. Franklin W. White, of Boston. (Applause.)

OBSERVATIONS ON THE HEALING OF GASTRIC ULCER*

BY FRANKLIN W. WHITE, M.D.†

THE life history of gastric ulcer is interesting; we all know its intermittent course. In studying such cases many important questions arise. What happens in the intermission of symptoms? Does the ulcer heal or is it merely quiescent? How long does healing take, and what relation does it bear to the age of the patient, the size of the ulcer and the duration of symptoms? What are the risks of medical study and treatment? How often will cancer be overlooked at the beginning of treatment, and how often will it develop later? What are the end results of medical treatment? We will try to answer some of these questions. They have practical importance because at present there is great diversity of opinion on the best treatment of gastric ulcer.

Many surgeons and some physicians advise resection only, many advise either resection or gastro-enterostomy. Others are content with gastro-enterostomy as a routine, and an increasing group of physicians are reporting satisfactory results with medical treatment.

This paper deals only with gastric, not duodenal, ulcer; it is important for various reasons to consider these two kinds of ulcers separately, and we shall have some special things to say about lesser curvature ulcers. The paper is a record of observations on the healing and the immediate and late results of medical treatment, and we shall pass briefly over the details of the medical treatment itself.

Fifty private patients with gastric ulcer have been under observation during the last six or eight years. The work was begun at a time when the accepted treatment of gastric ulcer was surgical and medical treatment was considered risky or doubtful. The results have been so interesting that out of them has grown a definite plan of medical treatment of gastric ulcer which has proved satisfactory.

THE USE OF THE ROENTGEN RAY

The Roentgen ray has great value in the diagnosis of gastric ulcer, and we have also found it very useful in watching the healing of the ulcer and in judging the immediate results of medical treatment. This is very easy on lesser curvature ulcers where the lesion is clearly seen in profile. It is much less definite in pyloric or duodenal ulcers. In lesser curvature ulcers under treatment Roentgen examinations at in-

*Read at the annual meeting of the Massachusetts Medical Society, June 11, 1929.

†White—Visiting Physician, Boston City Hospital. For record and address of author see "This Week's Issue," page 1116.

tervals of two or three weeks show that the deformity may steadily change and disappear, the lips of the ulcer may flatten, the deep crater fill and smooth over, overactive peristalsis become normal and pyloric spasm lessen and disappear. It is very important to remember that these changes coincide as a rule with clinical improvement and loss of symptoms. These changes may be surprisingly rapid even in large gastric ulcers, and all traces of extensive deformity may be gone in from four to six weeks.

We have included a series of Roentgen pictures* of seven of our lesser curvature ulcers under medical treatment in order to illustrate certain points. They are worthy of careful study. You will note the gradual disappearance of the features of the ulcer deformity referred to above, which has taken place during the cessation of symptoms.

The rapidity of disappearance of the ulcer varies from Case IV in one week to Case III in six months. In Case I the four cuts show the details of the progress of healing over a period of six months. Case II is an example of a large ulcer (crater 23 mm.) in a woman aged 53 which rapidly healed within six weeks. Case III is an example of a large ulcer (crater 30 mm.) in a man of 63 which healed slowly. Cases IV and V represent small ulcers and Cases I, VI and VII ulcers of medium size. Case V died at the age of 70 of arteriosclerosis, ten years after successful ulcer treatment; autopsy showed a normal stomach.

THE RISKS OF MEDICAL STUDY AND TREATMENT

When a suspected ulcer comes for examination and treatment we have to think of the possibility of cancer. Is it present? Will it develop later? The first question is far more urgent and important than the second.

Accurate diagnosis at the start is the most vital single factor in the treatment. The clinician and the Roentgenologist have great responsibility in deciding between gastric ulcer and cancer. It is often said that there is no sure way to tell, and it is true that no one factor in the diagnosis is entirely characteristic; not the history whether long or short or of a certain type, not the gastric acidity whether high or low, not the presence or absence of blood in the gastric contents and feces, not the kind of X-ray deformity or gastric peristalsis or spasm or dilatation or stasis, but if we take these factors *all together* a very accurate diagnosis is possible in nearly all cases. This is especially true if we watch the early result of medical treatment during a *probation period* of several weeks of intensive study. This is not a serious delay. We note the effect of treatment on the symptoms, on the blood in the feces, and especially on the X-ray deformity in the lesser curvature cases. The gastric ulcer usually shows definite and often striking signs of improvement in a short time. The gastric cancer is unlikely to change much.

This careful study soon clears up the diagnosis in most cases. We have never found a gastric deformity which rapidly improved in a malignant ulcer; this is a more reliable guide than changes in symptoms.

As a final safeguard, cases which are still in doubt after this intense study are best explored. In this way we have had no serious mistakes and no regrets. Many ulcers have cleared up rapidly under medical treatment. In doubtful cases some cancers have been excised, some ulcers have been excised (perhaps needlessly) but as far as we know no cancer has been left in which should have been taken out. Lahey has noted the fact that this preliminary medical study makes it much easier for the surgeon to know what operation to perform in the doubtful cases which he explores.

SELECTION OF CASES

In selecting cases for prolonged medical treatment we use the same rules which apply to duodenal ulcer. We avoid those with serious complications; perforation, recurrent hemorrhage or marked pylorie obstruction which does not yield, and those who for financial or social reasons or lack of intelligence are unable to carry it out. These latter are very few indeed in private practice.

OBJECTIONS TO X-RAY EVIDENCE OF HEALING

It has been said that the appearance of ulcer healing may be an artifact, that the crater may fill with food or mucus and seem to disappear when actually present, also that later films may be taken at another angle and fail to show an actual ulcer in profile. These objections seem unimportant and easily overcome by care in examination and interpretation. Lesser curvature ulcer gives a very definite picture as a rule and in all our re-examinations a series of six or eight films were taken at slightly different angles. In two cases the X-ray evidence of healing was verified by operation at the end of three or four weeks of treatment. The X-ray deformity had disappeared and the ulcer was found entirely healed at operation. Crohn, Richards and others report similar cases.

ULCER HEALING IN RELATION TO AGE OF PATIENT, SIZE OF ULCER AND DURATION OF SYMPTOMS

A study of our cases shows good evidence that gastric ulcer actually heals as a rule in the intermission of symptoms, and that healing may be very rapid, within a month or six weeks; this is true even of large lesser curvature ulcers. The rapidity of healing varied greatly from one week to six months. We were encouraged to continue medical treatment in the slower cases by the great clinical improvement and marked gain

*We wish to acknowledge our indebtedness to Dr. L. B. Morrison of Boston, who made one x-ray examination in three of these cases, and to Dr. E. L. Davis of Springfield, who made one examination in one case.

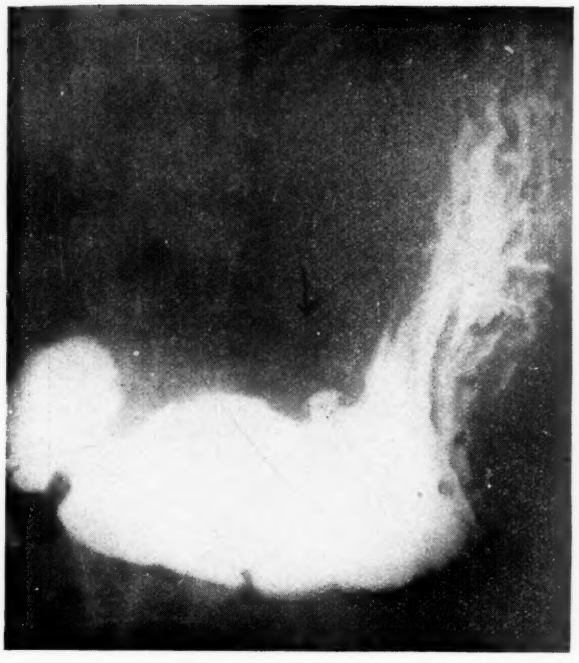

FIG. 1. Case I. Man, 45, symptoms 2 years. Medium sized gastric ulcer at the beginning of medical treatment.

FIG. 2. Case I. Five days later, slight change. Ulcer a little flatter, crater less deep.

FIG. 3. Case I. One monta later, marked change. Ulcer deformity almost gone, a trace of the crater remains.

FIG. 4. Case I. Six months later. Ulcer deformity entirely gone, patient well for 5 months (and has continued well for 5 years).

FIG. 5. Case II. Woman, 53, symptoms 2 months. Large gastric ulcer (crater about 25 mm. diameter) at the beginning of medical treatment.

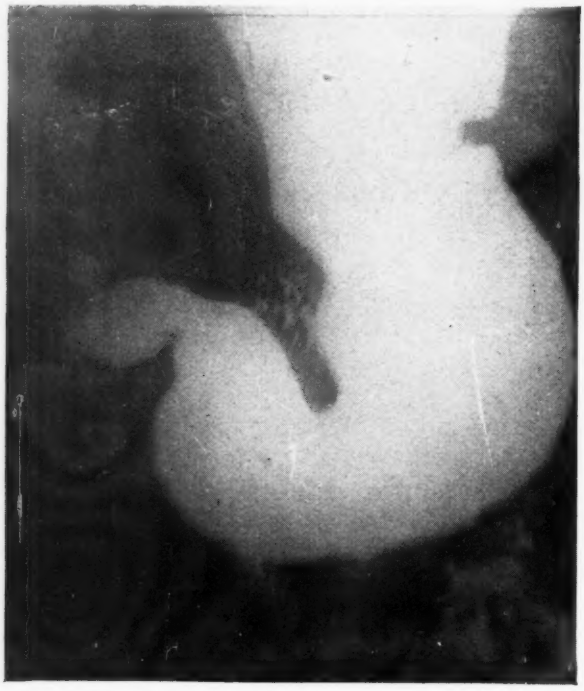

FIG. 6. Case II. One month later. Ulcer deformity gone, small incisura opposite.

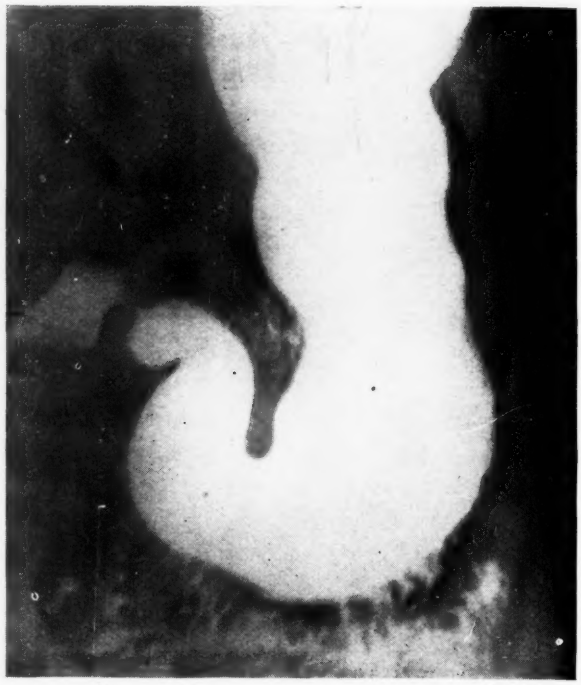

FIG. 7. Case II. One and a half months later. Deformity and incisura gone, lesser curvature flexible. (Has continued well 3 years.)

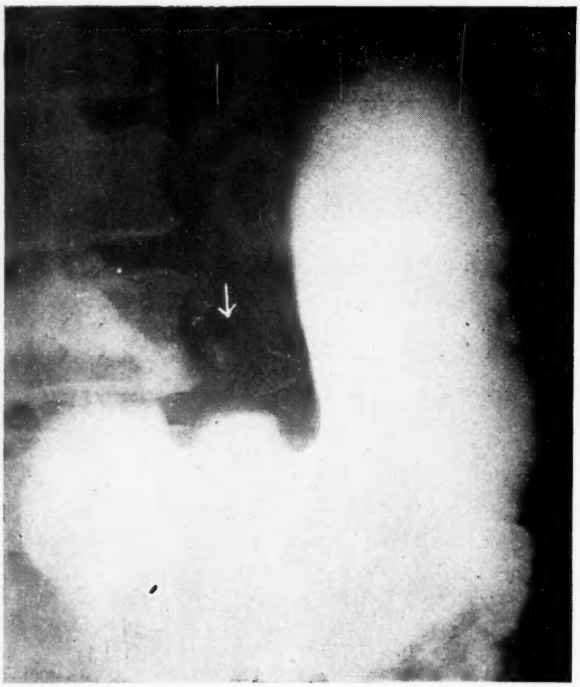

FIG. 8. Case III. Man, 63, symptoms 3 years. Large gastric ulcer (crater about 30 mm. diameter) at the beginning of medical treatment.

FIG. 9. Case III. Three months later, symptom free, gained 20 pounds, ulcer very much smaller.

FIG. 11. Case IV. Man, 50, symptoms 3 years. Small gastric ulcer at the beginning of medical treatment.

FIG. 10. Case III. Six months later, gained 35 pounds, ulcer deformity gone. (Has continued well 3 years.)

FIG. 12. Case IV. *One week* later, ulcer deformity gone. Symptom free. (Has continued well 4 years except for short recurrence.)

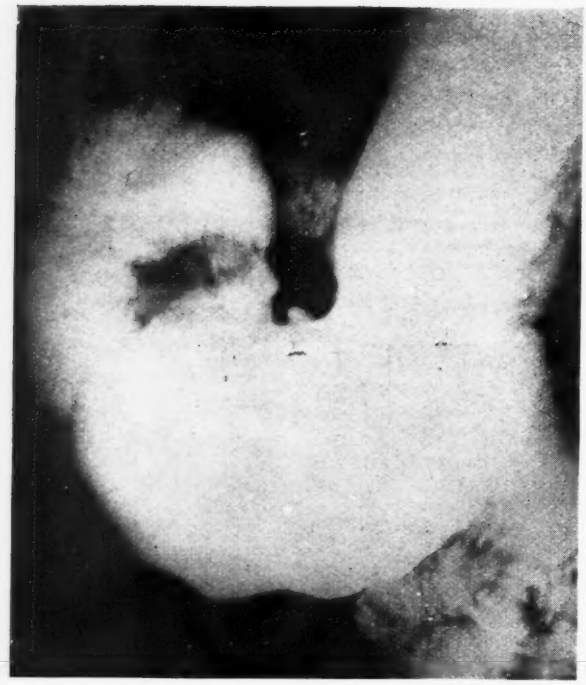

FIG. 13. Case V. Man, 60, symptoms 27 years. Small gastric ulcer at the beginning of medical treatment.

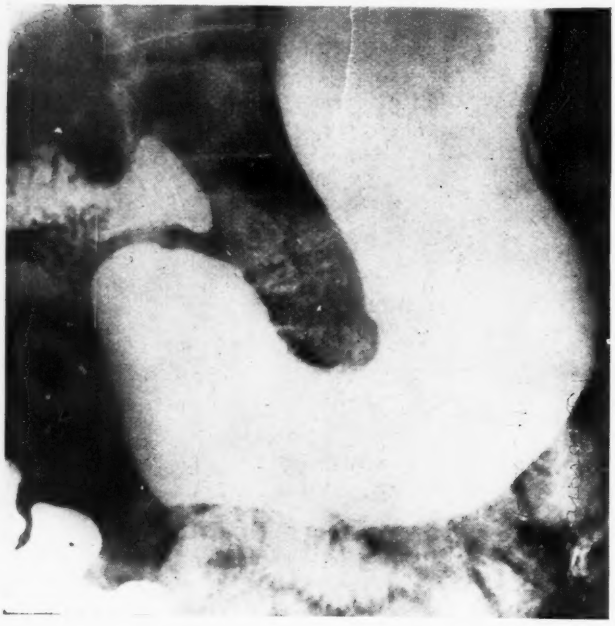

FIG. 14. Case V. Two months later, indigestion almost gone. Ulcer deformity entirely gone. (Continued well 10 years. Died aged 70, arteriosclerosis. Autopsy showed normal stomach.)

FIG. 15. Case VI. Man, 38, symptoms 6 years. Medium sized gastric ulcer at beginning of medical treatment.

FIG. 16. Case VI. Three months later, symptom free. Ulcer deformity gone. (Continued well 3½ years except for one brief, mild recurrence.)

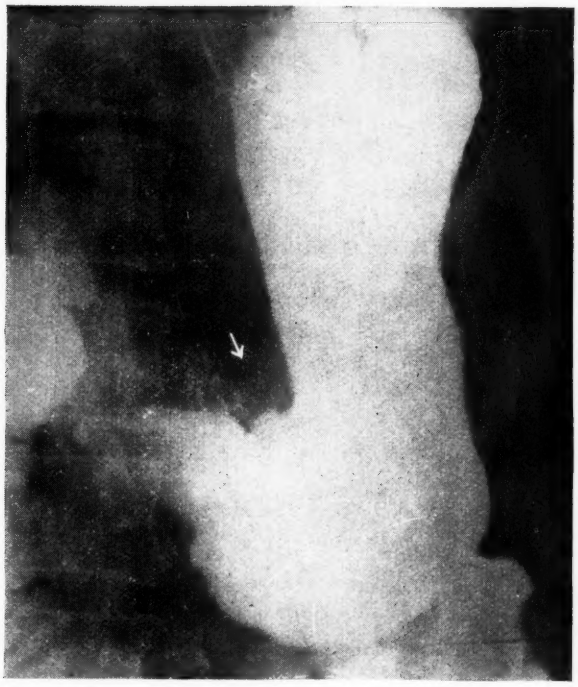

FIG. 17. Case VII. Woman, 65, symptoms 10 years. Medium sized gastric ulcer at beginning of medical treatment.

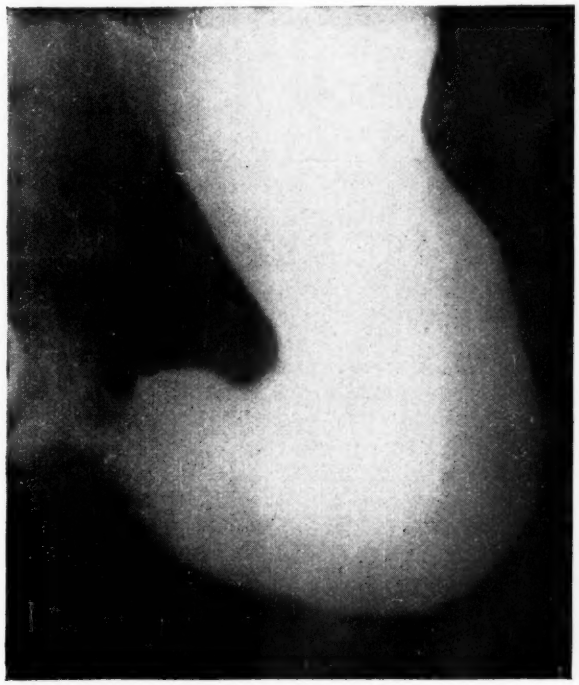

FIG. 18. Case VII. Five months later ulcer deformity gone (few symptoms, has also coronary sclerosis and hypertension, no ulcer recurrence in 3 years).

in weight in spite of the slow disappearance of the ulcer crater.

The *age of the patient* in our series has not especially influenced the healing. Most of the cases were 40-60 years old and the healing was much the same whether the age was near 40 or near 60. Two cases over 60 showed slow healing, both were large ulcers. Crohn has found that younger patients showed the greatest tendency to heal and that those over 45 healed with difficulty.

The *size of the ulcer* had little effect on the rate of healing. Most of our lesser curvature ulcers were of small or medium size with a crater 15-20 mm. in diameter (less than a 25 cent piece). The size of the crater has been approximately estimated from the films making due allowances for some magnification. Some of the largest ulcers with craters 25-30 mm. in diameter (about the size of a quarter) entirely disappeared within one month. The relation to the size of the ulcer to the possible development of cancer will be considered later.

The *duration of symptoms* had little effect on the rapidity of healing. Ulcers with four year symptoms healed just as fast as those with two months symptoms. Possibly some difference might show in a larger series of cases. Crohn found that the shorter the symptoms and the longer the intermissions the better the chance of healing.

We have found no instance of recurrent crater after its first disappearance, why I do not know. Possibly more frequent X-ray examination would have discovered some. Crohn, Büscher and others have reported several cases in which the ulcer crater recurred in the same place after six months to a year or more.

KIND OF TREATMENT

We will summarize this briefly. The details may be found in a recent paper by the author in the *American Journal of Medical Sciences*, May, 1927.

The treatment is usually begun with rest in bed for a week or two. We use frequent feedings and a modified Sippy diet with the addition of cereal gruels, low or moderate alkalies (from 50-150 grains per day) which is usually sufficient and avoids alkalosis and gastric irritation. We recognize the importance of the pyloric sphincter as a controlling factor in hyperacidity by regulating the emptying of the stomach and the regurgitation of alkaline duodenal juices into the stomach and use belladonna or atropin. The nervous system is quieted if necessary, tobacco reduced or omitted and focal infections eliminated. We tell the patient in advance that some treatment and supervision is needed for two years so he will have no illusions, and we continue treatment in the remission of symptoms. We consider it very important to *educate the patient* to manage his ulcer just as the patient is educated to manage diabetes.

END RESULTS OF MEDICAL TREATMENT

Our present-day accuracy of diagnosis of gastric ulcer without operation is over 90%. It is rare in a carefully studied case either to overlook an ulcer or to diagnose ulcer when it is not present. This is largely due to the addition of the Roentgen ray to our other methods. On this basis a new set of medical ulcer statistics have been accumulating in the last ten years which have real value.

The table shows the end results of medical treatment in 50 private cases of gastric ulcer; all have been followed for three years or more, the large majority for five years or more, and about one fifth for eight or ten years (figures for 200 private cases of duodenal ulcer similarly followed are given for comparison). We appreciate that cessation of symptoms is not a cure and that late results of treatment are the only valuable ones, the later the better. We always remember that we are largely treating the local lesion, not "curing" the disease, but in the present state of our knowledge this seems the best we can do.

	50 Gastric Ulcer		200 Duodenal Ulcer
	Number of cases	Per cent. of cases	Per cent. of cases
Well	23	46	60
Better	15	30	20
No better	1	2	4
Late operation	7	14	14
Died (medical)	4	8	2

These patients have been followed with painstaking care during medical treatment, accurate data obtained in the patients who were operated upon after medical treatment, and of the four who died autopsies were obtained in two and the abdomen was explored at operation in one. Nearly one-half (46%) are well, about three-fourths (76%) are either well or better. This group of patients who are "better" consist largely of those without complications and with long intermissions and mild recurrences who are unwilling to consider an operation. The group of late operations after the failure of medical treatment is rather small (14%).

Of the four deaths only one was from an ulcer which probably became malignant. This was a large lesser curvature ulcer in an elderly man who was inoperable on account of serious heart disease. The second case was a man seen at the age of 60 with a lesser curvature ulcer which rapidly healed (Figs. 13 and 14) and who died at the age of 70 of arteriosclerosis, autopsy showed a normal stomach. The third case was a man seen at the age of 72 whose gastric ulcer healed in a few months and who died at the age of 82 with cancer of the prostate. The fourth case was a man aged 58 whose ulcer healed rapidly and who died three years later following an operation for diverticulitis of the sigmoid. The

stomach was found normal at four X-ray examinations during these three years and was considered normal at operation.

Of the seven late operations after unsuccessful medical treatment four were done for persistent symptoms, two for pyloric obstruction and one for recurrent hemorrhage.

THE RELATION OF GASTRIC ULCER TO CANCER IN OUR SERIES

If cancer develops upon a previous ulcer it should be found either in the cases which have continued on medical treatment, or in the cases sent to the surgeon. It should appear in the material removed at resection, or should develop after gastro-enterostomy.

In our group of 50 cases we have one probable cancer developing in the 42 cases continued on medical treatment, most of them for five years or over, namely, 2% of the group. There were eight late operated cases. In six gastro-enterostomies no cancer has developed after four to eight years, in two late resections no cancer was found.

Lahey in 1927 reported 38 cases of gastric ulcer on medical treatment for three years with no development of cancer. Richards has recently reported 14 cases on medical treatment for two years with no cancer. Crohn in observing several hundred cases treated by both medical and surgical means could only recall one proved case in which malignant degeneration could be said to have supervened on a supposedly benign ulcer.

In addition during the period of this study 10 cases of gastric ulcer were sent to the surgeon at the start instead of carrying out medical treatment and three cases were resected and in seven gastro-enterostomy was done. No ulcers with malignant degeneration were found in the resected material and none of the gastro-enterostomy cases have developed cancer after periods of three to nine years.

The effect of gastro-enterostomy on a large gastric ulcer is sometimes amazing. Two of the largest and most massive gastric ulcers I have ever seen have entirely disappeared since operation and the patients are at present well and strong three and seven years after. These ulcers were so large and with induration so extensive that the surgeon felt sure at the time of operation that he was dealing with well developed cancer and only left them in because he did not dare to take them out.

Crohn in a surgical follow up of a large series of operated ulcer cases could recall only one instance in which cancer developed several years after a gastro-enterostomy for a benign gastric ulcer.

Our series with only one probable cancer does not permit us to say much about the size of the ulcer in relation to the development of cancer. Figures from the Mayo Clinic recently reviewed by Alvarez and McCarthy indicate that there are nine chances out of ten that an ulcer is benign if the crater is smaller than a quarter (23 mm.) and 14 out of 15 chances if the crater is smaller than a dime (18 mm.). Alvarez believes that if an ulcer is wrongly called benign the mistakes are usually in those with a crater over 30 mm., gastric ulcer being rare with craters over a quarter in size and gastric cancer being rare with craters less than a quarter in size.

We have felt very doubtful about accepting McCarthy's estimates of the incidence of cancer on ulcer for the reasons given in this paper, but it is interesting to note that nearly all our lesser curvature ulcers had craters which were smaller than a quarter (approximately estimated from X-ray films) only two were equal to or larger than a quarter; one 23 mm. and one 30 mm. in diameter. Both these ulcers cleared up, one promptly and one slowly, but both completely under medical treatment.

HISTORICAL

When McCarthy of the Mayo Clinic about twenty years ago reported that in a large series of resected gastric ulcer 68% showed areas of malignant degeneration it produced a tremendous effect because it seemed as if it was equivalent to saying that if these ulcer cases had been treated medically nearly 70% would have developed cancer and died, the conclusion seemed natural that since the mortality of resection was only 5-10% all these gastric ulcers *must* be treated surgically. This meant no medical treatment except to prepare for operation. Surgery dominated the field.

Gradually a change has occurred. Clinicians generally have raised their voices, gradually becoming more and more emphatic in dissent from the idea that over two-thirds of their gastric ulcers became malignant. Roentgenologists have joined the clinicians. Our guest today, Dr. Lewis Gregory Cole of New York, is a prominent dissenter. Many competent pathologists have disagreed entirely with Dr. McCarthy. Every man is impressed by the experience in his own locality. Dr. F. B. Mallory, the pathologist at the Boston City Hospital, says, "We have ulcerating cancers, but I have never seen a cancer originating on peptic ulcer, that is a chronic ulcer with cancer nests in the border or base." Dr. James H. Wright, pathologist at the Massachusetts General Hospital for many years, said, "Cancer developing on chronic ulcer of the stomach is very rare, that is cancer found in only one portion of a chronic ulcer, I have seen only a few, perhaps three or four cases in my life."

Finally many of our leading surgeons have been convinced that malignant degeneration of gastric ulcer is rather rare. This was clearly evident from recent discussions on gastric surgery in this city within the last six months by such men as David Cheever, D. F. Jones, F. B. Lund, F. H. Lahey and I. J. Walker. The

willingness of a surgeon to do a gastro-enterostomy in gastric ulcer throws light on his opinion of the frequency and importance of cancer development.

A strong impression has grown up that nearly all cancers supposed to come from ulcer were cancer at the start.

Our experience entirely disagrees with that of McCarthy, and though the group is small the per cent. ought to hold. Instead of finding 34 cancers developing in this group of 50 cases we have found only one possible case after a painstaking search. This experience makes us believe that there is a safe and reasonable medical treatment for gastric ulcer just as there is for duodenal ulcer, and if the cases are very carefully chosen and very carefully followed especially at the start, medical treatment need *not* be dominated by the fear of cancer.

SUMMARY

The Roentgen ray has proved very useful in watching the healing of lesser curvature ulcers and in judging the immediate effects of medical treatment. Gastric ulcer commonly heals and disappears in the intermission of symptoms. The ulcer deformity may entirely disappear in a week or two in some cases, and within four to six weeks in others even in large ulcers. In other cases the healing requires four to six months.

In our series the time of healing was not much affected by the age of the patient, the size of the ulcer or the duration of symptoms.

In diagnosis and treatment we try to be as sure as possible at the start that the lesion is ulcer not cancer, and then try medical treatment just as in duodenal ulcer. Surgery is not the primary treatment. We watch the immediate results of treatment with the greatest care by frequent examinations. Lesser curvature ulcers are much easier to watch with the X-ray than any others and yield remarkably well to medical treatment. A persistent follow up for three to nine years of 50 cases of gastric ulcer treated medically including those which had late operations, discovered only one case of probable development of cancer on (inoperable) ulcer, namely, 2% for the series. This low figure may be due to the fact that where the size of the ulcer could be estimated the majority were found to be of small or medium size. Our experience is that medical treatment of gastric ulcer is safe and reasonable if the cases are very carefully chosen and very carefully followed.

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CHAIRMAN MILLER: The paper is now open for discussion. The person appointed to discuss the paper is Dr. Sara M. Jordan, of Boston.

DISCUSSION

DR. SARA M. JORDAN, Boston: *Mr. Chairman, Ladies and Gentlemen of the Section*—I feel personally very grateful to Dr. White for his very instructive report on these fifty cases of gastric ulcer. It is always gratifying to be in accord with one whose judgment we respect so much. About five years ago Dr. Lahey became impressed and disturbed by the fact that many of the borderline cases of gastric lesions which were not frankly malignant, when resected, proved to be benign ulcers. He had seen certain ulcers, certain gastric lesions heal as evidenced by X-ray films, and he felt that some of these cases which were doubtful should not have been operated upon because the risk was too great to the patient, and that if possible we should adopt a policy for trying to find out which cases are ulcers and which are cancers.

At the Sippy Clinic, attention has been paid to the medical treatment of the gastric lesions and the observation of relief of symptoms, of occult blood in the stools and of the X-ray defect. We made it our policy to put all cases which were not frankly malignant—that is, very definitely so by X-ray, by gross irregularity of the contour of the stomach, and impairment of the tone of the stomach, by a suggestive history and by the presence and persistence of occult blood in the stools; those we classified as frankly malignant—on ulcer management, on careful ulcer management, and watched them for a period of ten days. That period has varied with different patients from ten days to four weeks, in one case, but it averages between ten days and two weeks.

In a study of 105 lesions of the stomach during the past few years, we have come to the following conclusions, that if we put out of consideration gastritis and the unusual tumors like myomata and lipomata and polypi, we may divide all gastric lesions into three classes: The frankly malignant, the frank ulcer, which must however be proved ulcer, and the questionable lesion, ulcer or carcinoma.

The study of these 105 cases has shown us that in this group there were at once fifty cases which were immediately diagnosable as carcinoma, that there were fifteen which were immediately diagnosable as ulcer, but which were also proved to be ulcer, and tractable ulcer, and forty which were at first doubtful. Of these forty, twenty-two were proved by treatment to be ulcer and were proved healed. Eight were considered unhealable, intractable, and seven of these have been operated upon. One

refused operation because he feels well. Nine were proved to be carcinoma and were operated upon, and one was inadequately controlled and was operated upon elsewhere and carcinoma was found.

The question of proving whether the lesion is carcinoma or ulcer is one very important phase of the subject, because the surgeon upon exploration with palpation and with visualization, as has been stressed by Dr. Lahey, is frequently unable to make the diagnosis. He cannot say even when he has the lesion between his fingers whether it is ulcer or carcinoma. If he has explored, he must operate and must resect, because he is afraid of leaving a malignant lesion. We feel there is no place for gastro-enterostomy in gastric lesions because if the ulcer is suspicious, it should be resected.

I will show a few X-ray films which demonstrate these points, but I also want to bring out this point: We feel we may not only prove whether the lesion is carcinoma or ulcer, but that we may also determine which lesion is a healable ulcer, a tractable ulcer and which is a chronic unhealable ulcer.

In an organ like the stomach which is susceptible to malignancy, we feel it is unsafe to leave a chronic ulcer. We, therefore, advise operation for two reasons, one, because we feel the lesion is suspicious of carcinoma and, two, because we feel that it will not be healed by medical treatment. In a short period of medical management, we believe we may determine which of these three eventualities will occur, whether the lesion will heal completely, whether it will only partly heal but remain a chronic ulcer, or whether it is a reasonably suspicious case of carcinoma.

(Slide) This first chart shows the history with drawings made to scale of X-ray films of a case of a woman of fifty-two years with a history of thirty-five years of indigestion, a typical ulcer distress. Notice also the size of the ulcer in the first drawing of November 26, 1924. Her free hydrochloric acid was low. She had occult blood in her stools. She had a large lesion of the lesser curvature. She was fifty-two. She had had a recent massive hemorrhage. All of those things were somewhat suggestive of carcinoma except for the long history. The long history was suggestive of the possibility that we might not be able to heal an ulcer of such long standing, if we assumed her symptoms were due to this lesion. In three days, you will notice the difference in the size of the defect. November 26 the first film was made; November 29, the second, and January 6, the third, and on July 14, 1927, two years and a half later, you see a perfectly normal stomach and the last film taken June 6, 1929, a few days ago, when the patient was perfectly well. This case gives evidence of the fact that even a large lesion in a person of middle age does heal. The patient is now well four years and seven months after the beginning of treatment.

(Slide) This next case is of a man of fifty-two who illustrated for us the fact that we may expect to detect the ulcer which will not heal. He came to the clinic in August last year with a three-year typical ulcer history. He had a high free hydrochloric acid. He had this larger lesion of the lesser curvature. He had occult blood in his stools which disappeared during treatment. His symptoms were completely relieved in a few weeks.

(Slide) The third slide is taken in a little different position. I see Dr. Cole in the audience, and I know he will notice that fact. But the film in the other position showed no evidence of the defect. In this position, however, we felt we could see a slight, a very minute area of rigidity here in this area, in the area of the ulcer. He was perfectly well. He insisted upon going home and we let him go with the advice to return in one month for another examination.

(Slide) He failed to return in one month, but returned in three months with the ulcer defect again visible, no symptoms, absolutely free of symptoms; but against his opinion, we persuaded him to be operated upon, and he had a chronic ulcer, an indurated ulcer, which showed some penetration into the pancreas. We felt quite sure that that ulcer would not have healed. We, therefore, feel that this is a good example of a case which by careful fluoroscopic and film examination can be shown to persist in the form of a very minute defect. The ones which heal well do not show this type of lesion.

(Slide) This shows the more difficult type, which Dr. White has already mentioned, of the lesion which is much more difficult to follow than the graphic ulcer of the lesser curvature, the lesion at the pylorus of the stomach. We are often confused in the interpretation of these films by spasm and we, therefore, at first put any lesion which is at all doubtful at the pylorus on medical treatment.

(Slide) This case, however, failed to show adequate improvement. There was a little relief of the spasm, but there was still the same defect at the pylorus. His occult blood disappeared from the stools but recurred. The symptoms were completely relieved, but in spite of that fact, we felt that he should be operated upon. He was operated upon and a very soft lesion of the pylorus was found. Dr. Lahey on palpating it said he could not have determined whether this was an ulcer or a carcinoma, but it proved to be an early adeno-carcinoma. This case is now alive, two and one-half years after operation.

(Slide) This is the same case after resection and he is well now.

We, therefore, feel that the three types of lesions may be distinguished by the presurgical criterion of a medical treatment. We think that the size of the lesion is unimportant. Some of

our largest lesions in old patients have healed well. The location is important. The lesions near the pylorus are more difficult to follow and more difficult to decide about. We feel that the age of the patient is entirely unimportant. Some of our oldest and some middle-aged patients have shown healed ulcers.

The duration of the symptoms is apparently also unimportant, except that the period of short symptoms is more suspicious of carcinoma, but with regard to the healing of the ulcer, apparently some of our oldest cases, the cases with the longest histories have healed as well as those with shorter histories.

The gastric lesion, we feel, must be proved to be healed, or it must be operated upon, and operation must be resection and not gastroenterostomy.

Thank you. (Applause.)

CHAIRMAN MILLER: It is found necessary to change the program in order to accommodate the other Section.

The next speaker is a man of distinction in his own community and well known throughout the country as an authority on X-ray. The list of his honors and distinctions I found in Who's Who is too long to quote. From experience of years ago, we know, to use the colloquial expression, he is a scrapper in his department. He enjoys a scrap more than anything else. This, however, I will leave to the audience to develop. The next speaker is Dr. Lewis Gregory Cole, who will give some X-ray views of ulcer of the stomach. Dr. Cole! (Applause.)

DR. LEWIS GREGORY COLE, New York City: *Mr. Chairman, Ladies and Gentlemen*—I seldom start a talk with an apology, but I believe that an apology or perhaps a word of explanation may not be amiss. The Chairman introduced me as a Roentgenologist. There is nothing in this paper I am going to give today that would in any way indicate I am a Roentgenologist and, therefore, I have gone a bit afield. In going afield, however, I wish to state briefly how I came to go afield. As a Roentgenologist, it has always been my practice to follow the cases to the operating table. As I went with these cases to the operating table, having stated the ulcer was at such-an-such a place on the stomach and of such-and-such a depth, I was surprised to see the minimum amount of pathology that could be observed and palpated at the operating table, and in some of the cases, had I not been there, the report would have come back that it was an erroneous diagnosis. If we showed ulcer half an inch deep near the lesser curvature and felt, or perhaps stated, or misstated that must have burrowed into an adjacent viscus, when we got there, we found that it hadn't burrowed into the pancreas or liver or any adjacent viscus; in fact, it wasn't even adherent to those. It was a surprise to me to see this depth of lesion that we saw in the X-ray and so little to show for it at the operating table where very often the surgeon showed me the courtesy of allowing me to scrub up and feel as well as see the stomach.

As a result of that, I began to study the pathology of gastric ulcer and from the pathology of gastric ulcer I began to wonder where these ulcers could be, in what anatomical structures they could exist. Then I studied the anatomy as described in anatomical textbooks and found that there was no place, and yet they were there. Then I began to study the actual pathological, and postmortem specimens, and that got me into the field of the comparative anatomy, the anatomy of the dead stomach as compared with the anatomy of the living stomach, and I found they were totally different things. In fact, the anatomy of the dead stomach, as described in any of the standard textbooks of anatomy, does not provide us with a space to put these ulcers on, but they are there, and if we go to look for the space, we will find it.

ANATOMICAL FACTORS PREDISPOSING TO CORPORIC ULCER*

BY LEWIS GREGORY COLE, M.D.†

ULCERS occur in three regions, namely (a) in the corpus, (b) in the antrum or pylorus and (c) in the cap, as shown in Fig. I. In each of these regions, the ulcer constitutes an entirely different problem and must be considered as a totally different entity from an ulcer in any other region. Facts concerning ulcers that occur in one region must not be applied to ulcers occurring in either of the other two regions.

Ulcers in each of these regions must be considered from various standpoints, namely (1) etiology (2) pathogenesis (3) pathology (4) diagnosis (5) course of the disease (6) results of medical treatment (7) results of surgical treatment, and (8) indications for medical or surgical treatment. As it is manifestly impossible in a communication of this brevity to consider all of these factors, we shall limit our discussion to a single type of ulcer. In other words, we shall deal only with corporic ulcer, and shall

*Read at the Annual Meeting of the Massachusetts Medical Society, June 11, 1929.

†Cole—Roentgenologist to seven New York hospitals. For record and address of author see "This Week's Issue," page 1116.

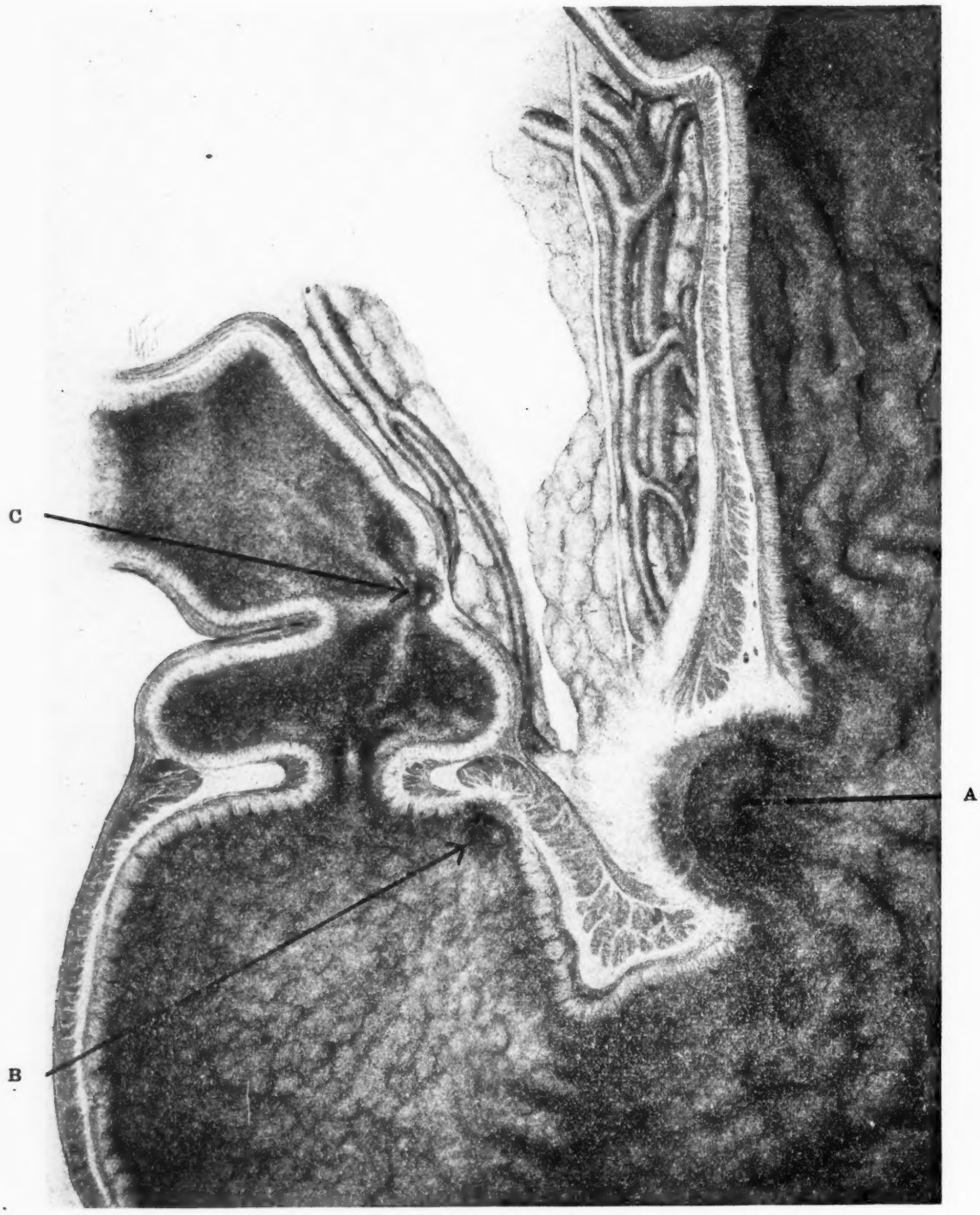

FIG. I. The three regional types of ulcer: corporic ulcer (A); ulcer involving the pyloric canal just proximal to the pyloric valve, termed prepyloric ulcer (B); and ulcer of the cap with cicatricial deformity of the cap (C).

confine the discussion to a single phase of the corporic ulcer problem, that is to certain anatomical factors predisposing to corporic ulcer.

There are two anatomical factors that predispose to corporic ulcer. One is a long, mucomembranous fold that projects into the lumen of the stomach and separates the corpus from the antrum (Fig. II). This long, mucomembranous

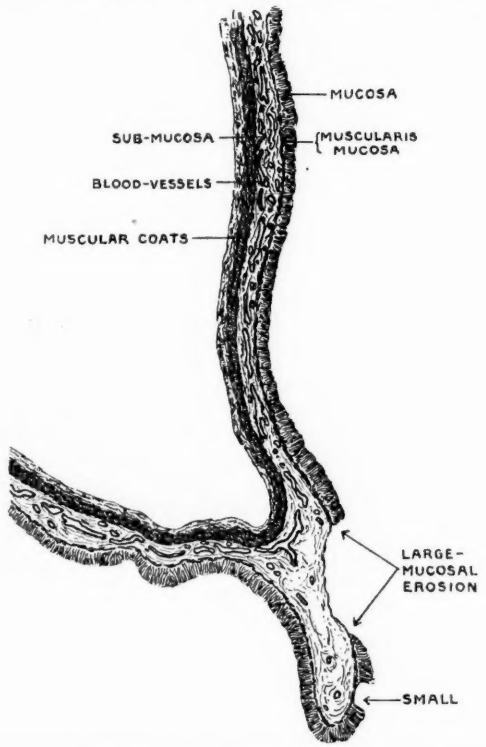

FIG. II. This drawing illustrates the muscular coat separated from the mucosal coat by submucosa. A large and a small mucosal ulcer are shown on the proximal surface of this mucomembranous fold (sulcus angularis) which projects into the lumen of the stomach, separating corpus and antrum.

fold has a limited blood supply derived from the portion of the gastric wall that Aschoff describes as the least vascular. Because this mucomembranous fold—or sulcus angularis—has already been considered in an article by the author in *Acta Radiologica**, it will not be discussed again at this time. Instead, we shall pass immediately to the other important anatomical factor predisposing to corporic ulcer, namely, to the anatomical characteristics of the left gastric artery and its branches.

Corporic ulcers occur only in that portion of the stomach supplied by the branches of the left gastric artery and for this reason our observations are confined to a study of this artery and its branches, particularly as they exist in the living, functioning, partly-filled stomach of a patient in the erect posture (Fig. III).

With the patient erect, the left gastric artery (Figs. III*i* and IV*d*), which is one of the branches of the ciliac axis, runs almost vertical and parallel with the right border of the corpus (Fig. III*a*). During its course, this left gastric artery gives off several branches that pass to the left (III*J* and IV*e*). These, in turn, divide into two branches each, one of which goes to the dorsal surface (IV*f*) of the stomach and the other to the ventral surface (IV*g*). The upper two or three branches of this artery are given off from a common stem (IVE). The lower or terminal branches supplying the distal end of the corpus are not derived from a common stem but directly from the left gastric artery.

The left gastric artery and all its branches are contained as a whole within a large, irregularly shaped, intraperitoneal space (Fig. III*h* and Fig. IV). This space is important in the ulcer problem because it is within this area that the majority of corporic ulcers develop. A comprehensive consideration of this space is essential to an understanding of the ulcer problem, but as such a discussion would require more time than is available it must be passed with only this meagre reference to its great significance.

The course of the left gastric artery (III*i* and and *l* and Fig. IV*d*) along the right border of corpus, within this intraperitoneal space, is behind and a trifle to the left of the left gastric vein (Fig. IV*c*) and the left vagus.

The upper two or three branches of the left gastric artery are exceedingly tortuous (III*j* and IV*e*) before their division into ventral and dorsal branches (Fig. IV*f* and *g*). The ventral and dorsal branches lie on the musculature of the stomach beneath the peritoneal coat that forms the anterior and posterior boundaries of the intraperitoneal gastric space. These ventral and dorsal branches do not enter the musculature at the right border, or so-called lesser curvature, but penetrate it at an angle (IV*h*) at a distance from the lesser curvature equal to approximately one-third of the circumference of the stomach (Fig.III*k* as observed through a window cut in the mucosa or Fig IV*i* as viewed in cross section).

After the ventral and dorsal branches of the left gastric artery have penetrated the musculature they enter the submucosa where they rapidly divide into a rich vascular network (III*k* and Fig. IV*i*). This division and anastomosis of the ventral and dorsal branches of the artery within the submucosa is best observed when the stomach is viewed from within after the mucosa has been dissected away (Fig. V). The manner in which these arteries penetrate the musculature at a considerable distance from the left border of the corpus, or the lesser curvature, and divide like crows' feet into a rich vascular network is shown in Fig. III*k* and V*e*. This penetration of the musculature deserves especial consideration. Through the semi-transparent peritoneum covering the ventral and dorsal surfaces of the dead stomach, the ventral and dorsal branches of the left gastric

*Acta Radiologica, Vol. VI, Fasc. 1-6, Nos. 29-34.

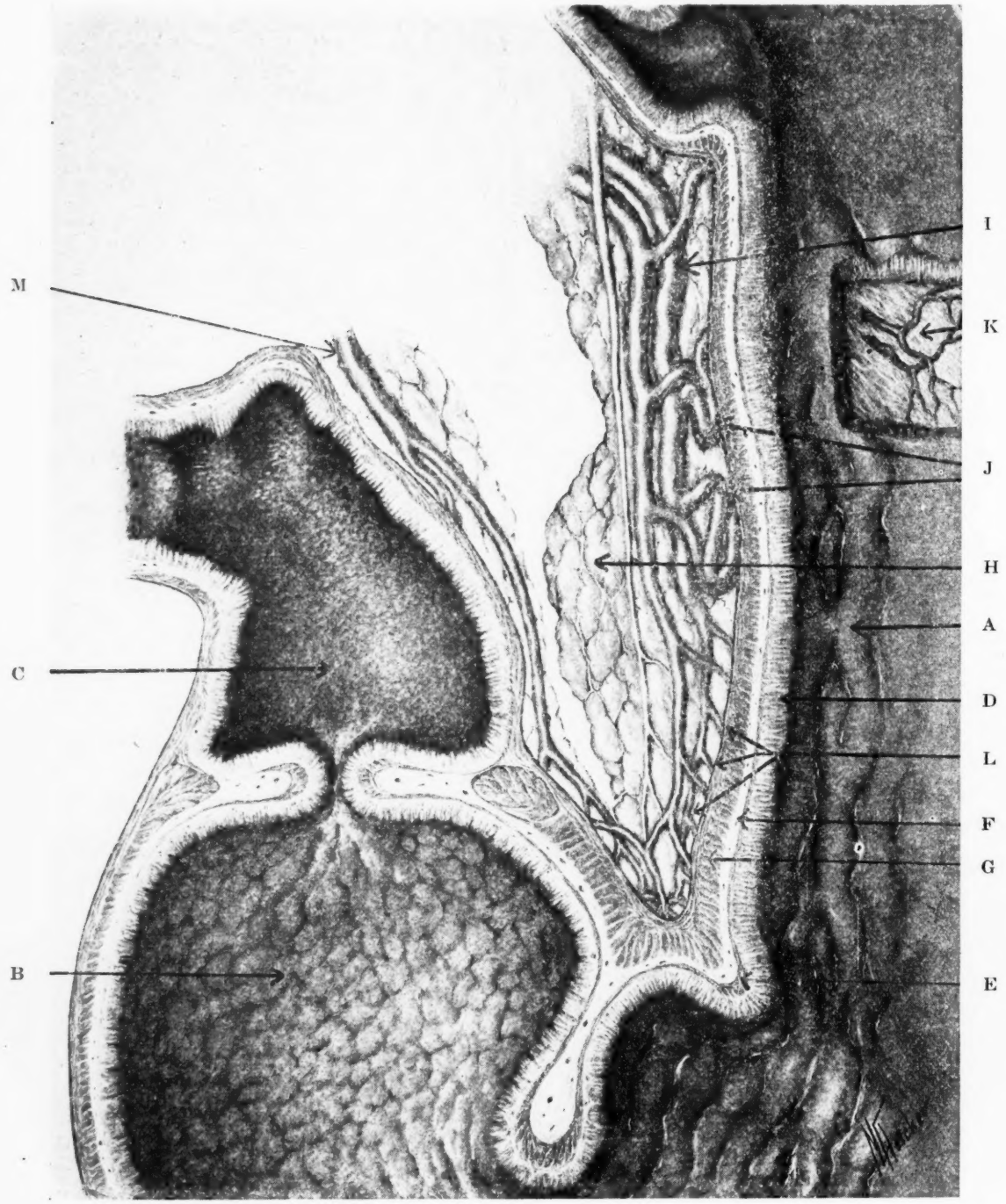

FIG. III. Ulcer-bearing area. Right border of the corpus (A); antrum (B); cap (C). The mucosa has characteristic appearance of each region. The various coats of gastric wall are shown as follows: mucosa (D); muscularis mucosa (E); submucosa (F); and muscular coat (G). The space (H) between the ventral and dorsal leaves of the peritoneum constitutes the intraperitoneal space containing the left and right vagus nerves, left gastric artery (I), and left gastric vein, which run parallel with right border of corpus. The upper two or three branches of left gastric artery are tortuous (J) dividing into two branches, one to ventral and other to dorsal surface, and penetrating muscular coat (K) to anastomose in manner shown. Lower distal branches (L) are singularly free of tortuosity, supplying stomach in incisura angularis region. Right gastric artery (M) passes downward, parallel with the left border of cap and antrum, and anastomoses with terminal branches of left gastric artery.

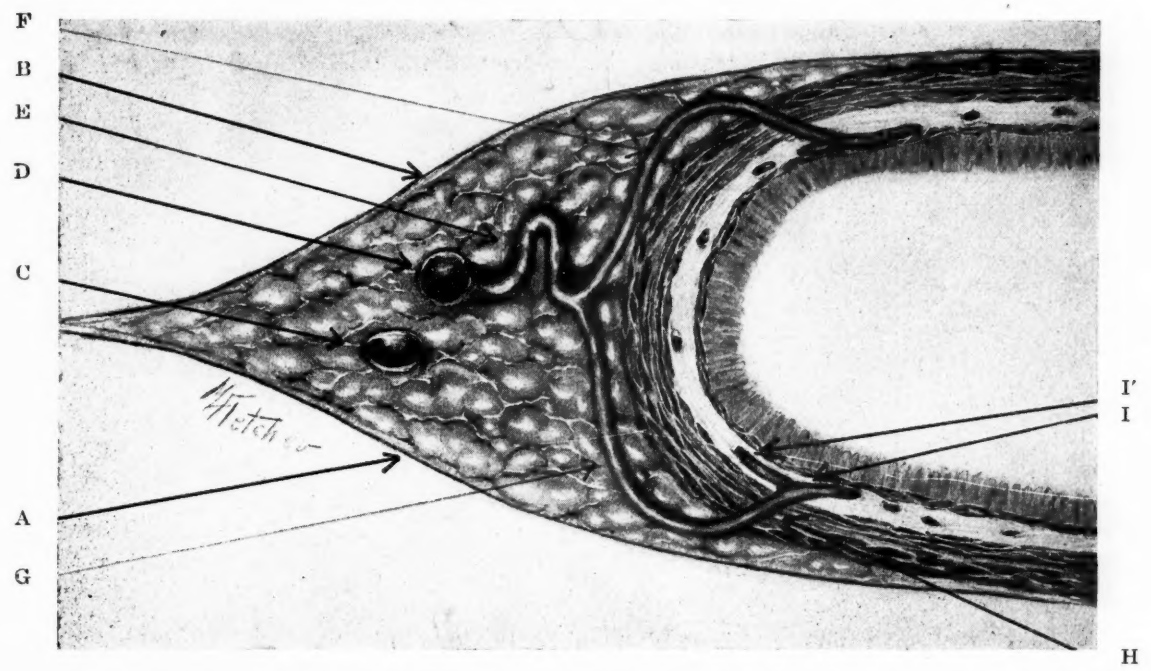

FIG. IV. Diagrammatic sketch of cross section of corpus, showing right side of corpus and intraperitoneal space between ventral (A) and dorsal (B) surfaces of peritoneum; left gastric vein (C); left gastric artery (D); tortuous branches of left gastric artery (E); dividing into dorsal (F) and ventral (G) branches, which perforate the musculature (H) and anastomose into rich plexus in submucosa (I and I').

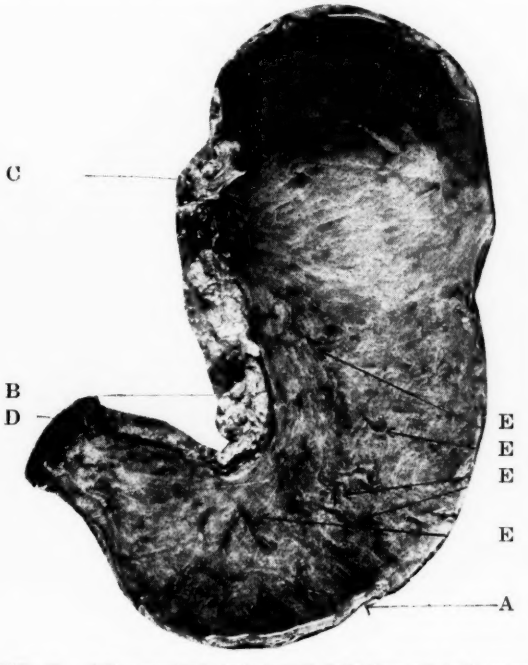

FIG. V. After ventral and dorsal branches of left gastric artery have penetrated musculature, they enter submucosa and rapidly divide into rich vascular network resembling crows' feet. This is best observed when stomach is viewed from within after mucosa has been dissected away:—greater curvature (A); lesser curvature (B); oesophagus (C); cap (D); arterial anastomosis (E) of penetrating branches of gastric artery.

artery may be observed. In the living, these may be seen to wriggle with each heart beat.

The terminal branches of the left gastric artery are distributed to the ventral and dorsal surfaces of the stomach near the lower end of the corpus in the region of the incisura angularis (Fig. VI*a*). These terminal branches are singularly free of the tortuosity that characterizes the two or three upper branches of the

FIG. VI. Untouched photograph of ventral surface of stomach showing terminal branches of left gastric artery (A) in region of incisura angularis. Left gastric artery is obscured by fat and areola tissue in intraperitoneal space.

artery that supply the upper portion of the corpus. This lack of tortuosity is undoubtedly due to the drag that occurs on these terminal arteries when, with the patient in the erect posture, the partly filled stomach sags.

With the patient in the supine posture—particularly when the stomach is empty or nearly empty—the greater curvature is drawn up by the contraction of the regular muscular coats of the stomach. In addition the greater curvature is supported by the transverse colon especially when the hepatic and splenic flexures drop into the sulcus on each side of the spine. Thus the drag or tension on these terminal branches of the left gastric artery is not noted in the cadaver which necessarily is observed in the supine posture. The tortuosity of the upper branches of the left gastric artery and the lack of tortuosity of the terminal branches is graphically shown in Fig. VII, an untouched photograph of a specimen prepared in a special manner that permits the dead stomach to retain the size, shape and position of the living, functioning stomach.

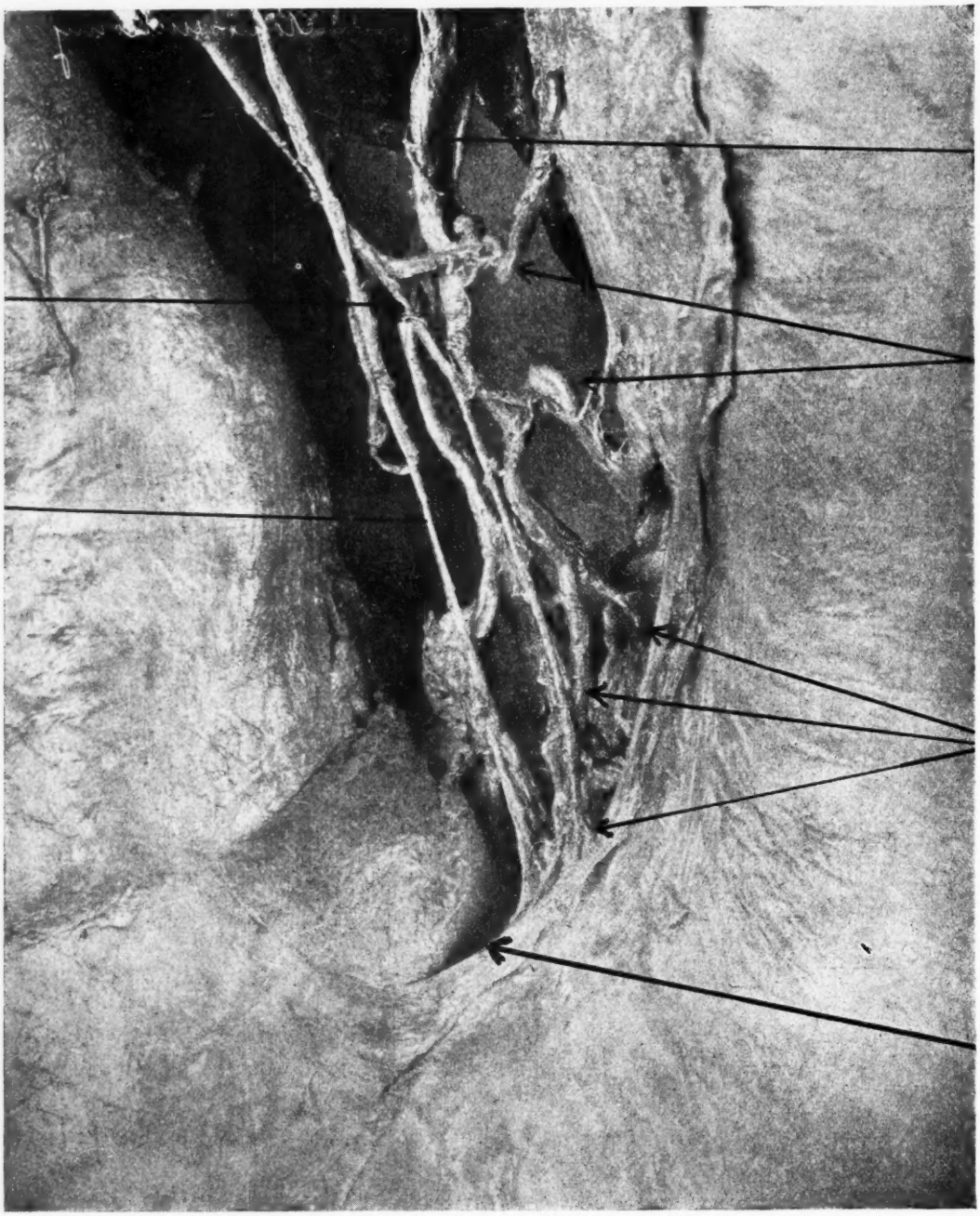

FIG. VII. The tortuosity of left gastric artery and lack of tortuosity of terminal branches is here graphically shown, an untouched photograph of a specimen prepared in a special manner to permit the dead stomach to retain the size, shape and position of the living, functioning stomach. Left pneumogastric nerve (A); left gastric vein (B); left gastric artery (C); upper two tortuous branches of left gastric artery (D); lower straight, or non-tortuous branches (E) terminating in the incisura angularis (F).

Photographs of specimens prepared in this

special manner show the characteristic distribution of the branches of the left gastric artery on the ventral and dorsal surface. Those to the dorsal surface (Fig. VIII*a*) are gen-

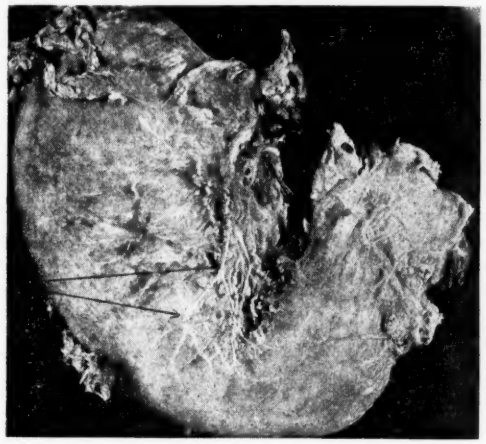

FIG. VIII. Untouched photograph of dorsal surface of a specially prepared specimen showing distribution of the branches of the left gastric artery (A) to dorsal surface, generally larger and more numerous than branches to ventral surface.

erally larger and more numerous than those to the ventral surface. When the fat and areola tissue are dissected away from within the intraperitoneal gastric space, these terminal branches are seen to pass to the ventral and

FIG. IX. When fat and areolar tissue are dissected away from intraperitoneal gastric space, the terminal branches of left gastric artery (A) are seen to pass to ventral and dorsal surfaces of stomach in fashion resembling division of reins of four-in-hand.

dorsal surfaces of the stomach in a fashion resembling the division of the reins of the four-in-hand (Fig. IX*a*). This is graphically seen if we observe the right border of the corpus with the pyloric end of the dead stomach cut away and with the lumen of the stomach filled with paraffine so that it maintains the shape of the living, functioning stomach (Fig. X). The space between the branches going to the ventral

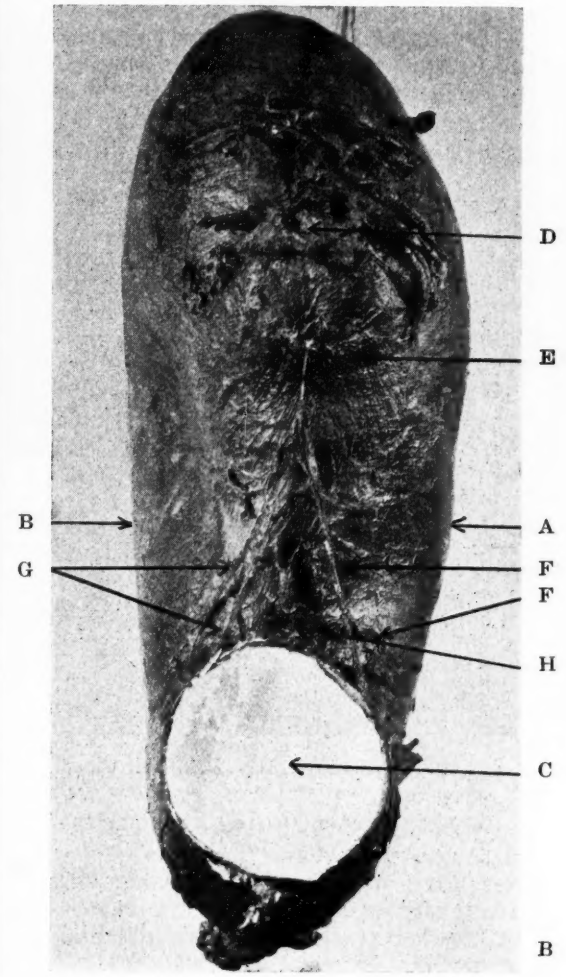

FIG. X. Right border of corpus with pyloric end of stomach cut away and with lumen of stomach filled with paraffine to maintain shape of living, functioning stomach:—ventral surface (A); dorsal surface (B); cut end of stomach (C); oesophagus (D); upper part of left gastric artery (E); ventral branches (F); dorsal branches (G); intraperitoneal space (H).

surface (X*f*) and dorsal surface (X*g*) of the stomach is bare of peritoneum (X*h*), and it is this space that is of great significance in the pathogenesis and pathology of corporic ulcers.

In a line drawing of the contour of the living, functioning stomach of a patient in the erect posture, dots have been placed to indicate the exact location of (a) corporic (b) prepyloric and (c) postpyloric or cap ulcers as observed roentgenographically by the author (Fig. XI). A line has also been drawn longitudinally through the corpus and antrum, midway between the greater and lesser curvatures. Before placing the dots, the locations of the pyloric valve and the oesophagus were carefully noted and measurements were taken from them so that each dot would indicate as nearly as possible the exact center of the ulcer. From a

survey of this schematic drawing, it becomes evident that many corporic, prepyloric and postpyloric ulcers occurred on the anterior or posterior wall instead of exactly on the lesser curvature. It will be observed that all the ulcers of the corpus and antrum occur on the lesser curvature side of the longitudinal line.

Returning to the actual photograph of a

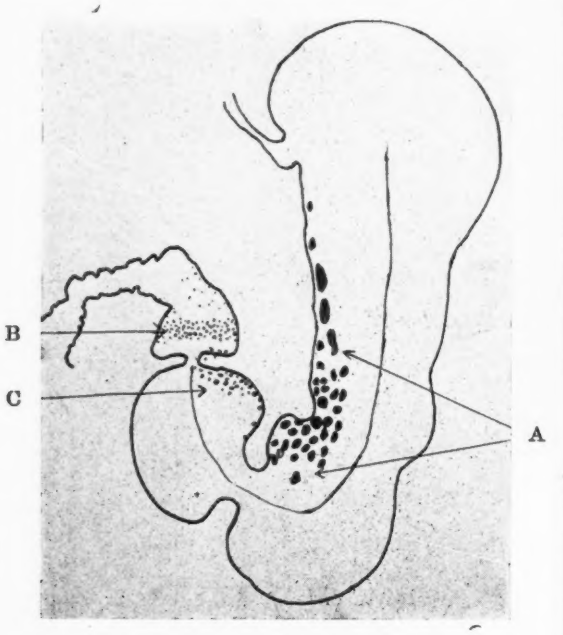

FIG. XI. Schematic drawing showing relative position and size of corporic (A); prepyloric (B) and postpyloric ulcers (C).

necropsy specimen which shows the distribution of the ventral and dorsal branches of the left gastric artery, particularly of the non-tortuous terminal branches, let us place an arrowhead at the point where each of the terminal branches penetrates the musculature (Fig. XII). We shall have, then, a schematic drawing showing the relative position and size of the corporic, prepyloric and postpyloric ulcers (Fig. XI) and a photograph (Fig. XII) of a necropsy specimen with arrowheads indicating the points where the terminal branches penetrate the musculature.

If we compare the schematic drawing with the actual photograph of the necropsy specimen (particularly if we superimpose one upon the other) a close coincidence between the dots and arrowheads at once becomes apparent. In other words, the arrowheads, indicating the points where the terminal branches of the left gastric artery penetrate the musculature, correspond so closely with the points of predilection for corporic ulcer that unquestionably there is a definite relationship between the two.

Going a step further and correlating this fact to the practical treatment of corporic ulcers, it will be recalled that the tension of the terminal branches of the left gastric artery is greatest with the patient in the erect posture, especially in the hyposthenic type of individual whose stomach tends to sag, and that, vice versa, the tension is least when the patient is in the supine

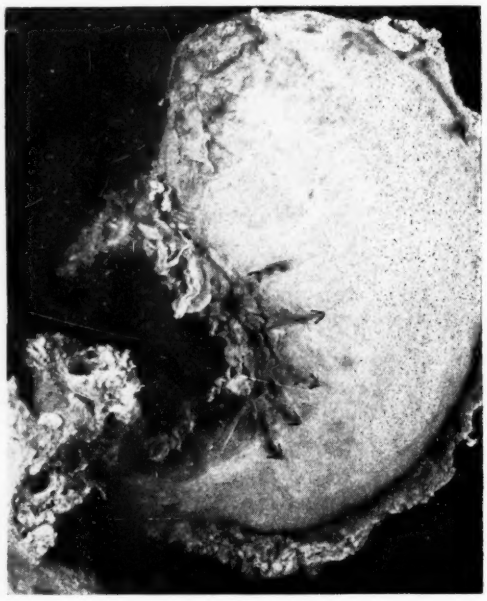

FIG. XII. Necropsy specimen with an arrowhead placed at the point where each terminal branch of left gastric artery penetrates musculature.

posture. This fact demonstrates the rationale of the rest in bed treatment of corporic ulcer, which has been employed hitherto purely on an empirical basis.

CHAIRMAN MILLER: Gentlemen, I have lived up to my agreement with the Physiotherapy Section and Dr. Cole has now finished so that there is plenty of time for discussion. While we are discussing his paper, I also expect you will take up the discussion of the paper preceding, that read by Dr. White, on "Observations on the Healing of Gastric Ulcer." It is a paper well worth more discussion than I allowed you before, but it can be very well carried on, and, in fact, might very well be carried on during the discussion of this last paper. Dr. Cole has challenged you, and I hope both medical and surgical men will see fit to discuss both papers freely, remembering, however, the five-minute period allowed you. You will be called to time by the Secretary.

DISCUSSION

DR. F. B. LUND, Boston: *Mr. President*, I was delighted with Dr. Cole's paper. I have long felt that cancer started in as a penetrative ulcer and very rarely developed from ulcer. I have seen so many cancers that started without history of ulcer and progressed very rapidly and were in different parts of the stomach from what Dr. Cole has explained as the ulcer area and perhaps as what we have known before as the ulcer area. A careful study and a care-

ful therapeutic test, a test of the results by the X-ray, are, therefore, completely justified and there is very little danger of cancer developing. I know how quickly an ulcer will heal after a gastro-enterostomy, and that was demonstrated to me a great many years ago in a woman with a cancer in whom a diagnosis of cancer of the pylorus had been made. She had just had a very large hemorrhage, therefore, she was in no condition for resection. Gastro-enterostomy was done in the presence of a great indurated mass which I couldn't tell whether it was cancer or ulcer. She improved very rapidly, but I wanted to be sure, so I opened her two weeks later and the whole thing had disappeared. Now that is certainly a rapid cure.

In regard to Dr. Jordan's discussion, I agree with it almost entirely, but I do not think that one ought to make the sweeping statement that gastro-enterostomy has no place in ulcers of the stomach or duodenum. I don't know whether Dr. Jordan referred simply to the lesser curvature.

DR. JORDAN: To the stomach only.

DR. LUND: In that I would almost agree with her, but I have seen cases in patients in poor condition with an ulcer adherent to the pancreas over an extensive area where a gastro-enterostomy was done and where a subsequent operation showed that the ulcer had been entirely cured. I would agree with her that resection almost always ought to be done in those cases.

In regard to ulcer at the duodenum. I have resected ulcers in the first part of the duodenum in one case and found it was a cancer, because it looked like it, but the most wicked ulcers of the duodenum, the largest and most indurated, extend down onto, I suppose I ought not to say the duodenum, because Dr. Cole says it isn't, but that is what we have always called it. You have to go down a long way to get them out and they do heal wonderfully after gastro-enterostomy. I am perfectly sure, as sure as I stand here, I don't care who says otherwise, that gastro-enterostomy is to be done in duodenal ulcers, except where you have to stop a bad hemorrhage, and where ulcers are unfavorably situated for excision, and which do not heal under medical treatment. I think our ideas have cleared very much on this subject by such work as has been done by both the speakers.

In regard to Dr. Cole's paper, I think I wouldn't say that ulcers never happen under the great curvature, because I have seen them, located them and cut them out, but not very often. (Applause.)

CHAIRMAN MILLER: Will someone else take up this discussion, please?

The next speaker on the program is one already known to you by sight, advice and reputation. I have great pleasure in introducing Dr. Lund who will announce his own title.

DR. F. B. LUND, Boston: I was asked to read a paper on something connected with the gastro-enteric tract and it seemed to me the thing foremost in my mind was in connection with the surgery of the gall-bladder and had to do with the medical treatment before and after operation and also with the matter of anesthesia in gall-bladder cases. Therefore, I have written this paper.

Seventeen years ago I reported 347 cases. I haven't counted up in the last seventeen years, but I thought I would take as a basis of this paper the cases that I have had since I joined the Carney Hospital staff, which was three years ago. They have been 84 in number. Of course, in statistics that has a disadvantage that one death more or less makes an awful difference, the fact to which Dr. White has alluded. I knew it was better to take the things of which I had good records and could tell you definitely, than to go farther back. I will now begin what I have to say.

THE IMPORTANCE OF MEDICAL CARE AND CONSULTATION IN CASES OF GALL BLADDER DISEASE. ALSO THE ADVANTAGE OF SPINAL ANESTHESIA IN OPERATIONS ON THE GALL BLADDER*

BY FRED B. LUND, M.D.†

THE surgery of the alimentary tract, as far as inflammatory disease is concerned, concerns chiefly the surgery of the appendix, gall bladder, stomach and intestine. By far the larger number of cases known, concern the appendix and gall bladder. The anatomical relations of the appendix are simple compared to those of the gall bladder, and the operation upon it has been fairly standardized. The gall bladder and common duct are related by position closely to the duodenum, kidney and stomach and complicated often by concomitant lesions of the stomach and pancreas. Gall bladder disease concerns patients of more advanced age, with its accompanying lesions of the heart, lungs, arterial system and kidneys, and requires perhaps more

*Read before a meeting of the Massachusetts Medical Society, Medical Section, June 11, 1929.

†Lund—Surgeon-in-Chief, Carney Hospital. For record and address of author see "This Week's Issue," page 1116.

judgment as to time and character of operation, more manipulative skill, than perhaps any other class of diseases the surgeon has to treat.

In general it may be said that the operations on the gall bladder are not quite as urgent as those on the appendix. Acute gall bladders, even, can usually wait till morning, for instance, without danger of perforation; but once in the writer's experience, which now comprises many hundreds of cases, perforation took place in an acute case, which was seen in the evening, during the same night. In the morning the tumor had disappeared and at operation most of the stones were found not in the gall bladder, but free in the abdominal cavity. Very acute gall bladders, then, are urgent, especially when gangrene threatens, but can usually wait a few hours, or one night if necessary. In subacute cases, one may well wait, and ought to wait for the attack to quiet down, especially if the patient is distended, hiccoughing or has myocarditis or a bronchitis; but here also one must be careful, and if steady improvement with fall of pulse and temperature does not take place, one should not wait. One may have a perforated gall bladder with an abscess which will not quiet down till it is drained, or one may have a gradual extension of infection with fat necrosis into the pancreas, both of which things have happened to me. It is easy to make rules, but rules have exceptions, judgment is not easy, and experience and the appearance of the patient tell a surgeon more about when to operate and when to wait than anything laid down in books. The history is the most important item in the diagnosis, but the physical examination and the look of the patient alone tell when a case is urgent. In regard to subacute and chronic cases in old fat people, and they are usually old and fat at the Carney Hospital, one should always wait long enough to study the patient, make an accurate estimate of the cardiac and renal condition, and if necessary give digitalis salt solution, glucose, etc., for several days, get the bowels well emptied and the general condition up to or above par. I have waited three weeks for hiccoughs to get well in an old man with a gall bladder way back under his liver close to the spine, and gotten away successfully with drainage and the removal of the stones. At the Carney Hospital we have made it a practice in all cases which are not urgent, to have a careful examination and treatment by the internist in charge. Most of the work on my cases has been done by Dr. Edward J. Denning and Dr. Louis F. Curran. The results of their pre-operative work has been to me extremely valuable and has been of especial help in elderly, obese and broken-down patients. I have asked Dr. Denning to contribute a few remarks on the importance of thorough pre-operative medical work on these cases, and following is his contribution to my paper:

"When a patient submits himself for operation, an internist should evaluate the patient's medical and mental condition, especially the condition of the cardio-renal system, because the functional efficiency of the heart and kidneys greatly affect the operative risk. The consultation with an internist will relieve the surgeon's responsibility in the event that the post-operative course of the case is attended by medical complications which may have been predicted prior to operation. The surgeon's skill may be frustrated by an unfortunate or too hasty acceptance of an unsuitable surgical case. In the future, the advance in surgery must be looked for in the knowledge gained from a more comprehensive pre-operative study of the patient. Operative mortality can be further reduced by a careful medical supervision before operation and by delaying the operation until the patient is in the most favorable condition. Surgery has been made safe for the patient—now let the patient be made safe for surgery.

"In emergency surgery, immediate operation is demanded and there may not be sufficient time for a thorough examination, but chronic cases can be subjected to thorough study. Not only diabetes, hyperthyroidism and enlarged prostate may be found, but also the complications which accompany anemia, acidosis, asthenia, obesity, inanition and dehydration. By careful history taking we may find a history of scarlet fever, rheumatic fever, typhoid fever, etc., all well known for their effect on the heart, blood vessels and kidneys. Low progressive changes in these organs may be present without symptoms, until functional tests suggest the presence of actual pathological change. Not infrequently in patients of forty-five (gall bladder age) we can elicit a history of dyspnea, palpitation, precordial and thoracic, pain on exertion, transient edema about ankles, insomnia, fatigability periods of depression, vertigo and a sense of fullness in the cranium. Unless we deduce from these symptoms of cardiac weakness or vasomotor instability, we are apt to precipitate our patients into circulatory embarrassment. Therefore we look for the following:

1. Size of Heart and retrosternal dullness, even to check up by x-ray if necessary.

2. Efficiency of heart muscle:

a. Particular attention to the character of the first sound at the apex; if the first sound diminishes or approaches in character the second sound, we can reasonably infer that the myocardium has suffered some change.

3. Irregular heart action.

a. In young people an extra systole is generally of no significance. Extra systoles are often found for the first time before operation in patients whose hearts are normal. This is undoubtedly due to the mental state. Irregularity in middle age or older may be of much significance and should be studied carefully for some time.

4. Tachycardia.

a. Due to psychic causes and should usually cause no apprehension. If it persists over a long period, it may indicate hyperthyroidism, tuberculosis or a focus of infection.

5. Pulse.

a. Note rhythm: volume and tension should be taken into consideration.

6. Blood pressure.

a. Diastolic reading is very important as this represents the pressure in the arteries between the heart beats. Just in proportion as this is persistently above the normal figure, it suggests permanent changes in the vessel walls and a continuing increased demand on the left ventricle. In those cases a retinal examination adds to the picture.

7. Kidneys.

a. Twenty-four hour urinalysis. If anything is found then B.U.N. Phthalein and Mosenthal tests before operation should be made.

8. Blood count.

a. Thorough blood examination should be done, not only white and differential, but red count and hemoglobin. In this way we may find occasionally a leukemia or pernicious anemia.

9. Effect of Fear.

a. Fear is a drawback to operation, therefore we try to prepare all patients for the anxieties, the discomfort and the ordeal, incident to the operation. If these are reduced as much as possible, we believe that the mortality will be lowered. As most gall bladder cases are not emergency operations, it is usually convenient for the surgeon to get an estimate of the patient's ability to stand the operation from an internist of experience, and not depend alone upon the man who referred the case or the house officer at the hospital where the operation is to be performed. The usual routine, blood pressure, urinalysis, complete blood is done in the laboratory and a thorough examination is made of the heart and lungs by the house officer in charge, to be followed by a painstaking thorough physical examination by the visiting internist.

"Our results in gall bladder cases in a great measure can be attributed to the fact that any remediable cardiac condition is prescribed for before operation and cardiac stimulation given until the patient is considered at least a fair risk. Most of the cardiac cases met with in gall bladder disease seem to be myocardial rather than endocardial—poor heart muscle rather than diseased endocardial lining, although some cases show auricular fibrillation after many years of good compensation of mitral disease. Our routine method is to give pill digitalis six grains as initial dose, followed by one and a half grains every four hours until heart sounds are better and stronger.

"A complete physical examination may eliminate many complications before rather than after operation. No longer are diabetics operated upon, only to find out later that they have had diabetes for years. Pernicious anemia is detected before rather than after operation.

"Most surgeons are willing to acknowledge, as they ought, that their knowledge of the heart is not to be compared with that of a good internist. This is quite different from twenty years ago. When one thinks how the efficiency of the surgeon has increased, it is only fair to admit that medicine has progressed also. The internist is now able to size up the heart better than he was at that time.

"Any hospital that uses its medical department early and often, not only before but after operation, presents less mortality than prevails in those in which the medical and surgical services act as almost separate units.

"If such importance can be attributed to preoperative medical consultations, even much more can be attributed to early and frequent postoperative consultations. We know that massive collapse of the lung is recognized more and more frequently now than formerly. We can detect this early and by its recognition and treatment, not only help but prevent this frequent complication. We detect emboli now that formerly were called pneumonia, emboli which when they occur early are not so fatal as pneumonia. Shock and hemorrhage have become extremely rare, but we still find as post-operative complications emboli, pneumonia, massive collapse of lungs, thrombophlebitis, acute myocardial failure, etc."

With the above I am in full agreement. Yet in our reliance upon the medical man we may go too far. I mean that the surgeon must follow his own cases before operation, and decide when and how long medical treatment should be continued. If a patient has a bad heart and kidneys, together with a perforation or abscess, medical treatment is going to do no good till the abscess has been drained, and what surgeon of experience has not waited in cases which grew worse while he did. We are back again with old Hippocrates:—"Judgment is difficult, the occasion urgent, experiment dangerous."

It is those cases in which the surgery must be done in spite of the patient's poor condition that we get our higher mortality, as will be illustrated by the fact that our mortality in cholecystostomy, into which class the desperate last-chance cases fall, is four times as high as that of the more technically complicated operation of cholecystectomy.

In July, 1898, we began to use spinal anesthesia in the cases where the heart or lungs were in bad shape. We have been gratified at our results. We have not, however, used spinal as

a routine in these cases, having employed it in ten cases, about half the cases done since we began to use it. In certain cases there has been a little anxiety due to the pallor of the patient and the slight fall of blood pressure. I understand that this fall of blood pressure is due to the relaxation of the vaso-constrictor muscles by the paralysis of the sympathetic, so that the patient for a time bleeds into his abdominal and other superficial veins. It has been easily controlled by lowering the head of the table. The use of preliminary morphine has helped and the proper employment of ephedrin has minimized our troubles of this sort. We employed first the novocain crystals, that is the so-called Spinocain, or Pitkin's solution, and have now returned to the novocain crystals which Dr. William A. Sheehan, associate anesthetist to the Carney, who has done most of our spinal and other anesthesia for us, has found a little more manageable. We have done only ten of our gall bladders with this solution and usually have been delighted with the abdominal relaxation and easy access to the parts. We have had only one failure and no mortality, not only in our gall bladder cases, but the others in which spinal anesthesia has been indicated, about fifty. The bugaboo that it was inhuman to operate on a conscious patient has long been squelched. My last spinal gall bladder told me the next day that she heard a lot of new stories during the operation which she was going to add to her repertoire.

The chief indications for spinal anesthesia to our minds are bronchitis or other respiratory trouble, and cardiac disease. In very bad cases we use a combination of local and gas, but the relaxation is inadequate, and a great contrast to the ease of spinal. However, we sometimes have to use it for safety's sake. The convalescence in the spinal cases as to vomiting after operation and early passing of gas is very satisfactory. Dr. Sheehan says they pass gas more easily because the sympathetics are shut off, leaving the vagus to act unopposed. I suppose that is the reason, but anyway they do it. The Trendelenberg position, I am also told, is essential when spinal anesthesia is used, to prevent cerebral anemia on account of dilation of the peripheral vessels. This does not interfere with ease of operating.

I can tell you a simple way to make a gall bladder operation easy. Don't put a pillow under the back to produce lordosis and stretch the abdominal muscles, a foolish procedure which was so much in vogue a few years ago that they even made operating tables with a cross bar for that purpose. I found out by forgetting to do it a few times that I had much better relaxation of the muscles and a whole lot better time.

I have, written down in my notes, the exact dose of crystals and ephedrin, and how to use them under all circumstances and what to do if there is trouble, but I am not going to tell them to you. When you do use spinal, get some one to do it who has made a study of it and don't learn how on your own patients, if you do not, as I do not, know all about it yourself.

In regard to the vexed question as to whether to remove the gall bladder or drain, a few words may be said. Each case must be decided on its own merits. Sometimes, as every one of experience knows, the removal of a gall bladder is ridiculously easy, sometimes it is difficult and dangerous. In cases of definite pathology it should be removed if it can be safely done. In cases of gall stones under the same circumstances it should be removed to prevent recurrence. But in cases of obese patients in poor condition where excision is so difficult as to add definitely to the risk, drainage should always be done. I have several patients living and well upon whom drainage has been done twice, who, I believe, would have died if I had attempted cholecystectomy. Cholecystectomy may be done later after a drainage, if symptoms persist, but your patient is alive anyway. It is usually not difficult in a given case to decide which procedure to choose. One may fairly put it, that in the simple, safe cases you are a fool if you don't do a cholecystectomy and in the difficult, dangerous ones you are a fool if you do. With the removal of gall bladders which look normal, are not thickened, and which empty under pressure, because of symptoms suggesting gall stones, for which exploration has been done, I have no sympathy whatever. How a man can have the conscience to expose his patient even to a slight danger I cannot conceive. Such a procedure certainly violates the oath of Hippocrates, the golden rule and every other law of God or man.

In regard to the x-ray, shadow pictures taken by the dye are a distinct aid, yet gall bladders which fill with the dye may be infected and require removal. They fill because the cystic duct is open. On the other hand, gall bladders which for some reason do not fill may not be pathological. In general, the dye may aid us in making up our mind. But the symptoms are of paramount importance. When the diagnosis is plain, I do not bother with the dye. What is the use when you can feel the gall bladder, or can get a definite history with local tenderness and spasm? In acute cases the delay is a serious matter. In my own mind the dye should not be used except in chronic cases where the diagnosis is in doubt. It is just one more nuisance for the patient and costs time and money.

One word about drainage in cholecystectomies, I always drain, because no matter how carefully or how easily the operation may have been accomplished, one is never certain that there may not be an escape of bile for the first two or three days, and I would prefer to have it run out into the dressing than into the abdominal cavity. This gush may be due to the fact that there is an

abnormal branch of the cystic duct or that my ligature may be loosened by the subsidence of the inflammation in edematous tissue. This drainage is done by means of a stab wound in order that the operative incision may be closed without drainage, or in case much bile has been spilt, may be drained merely by a bit of rubber tissue passed down to the peritoneum. I would run at full speed away from an operator who proposed to operate upon me, but said he never drained his cholecystectomies. The drain is left in six or seven days, and by twisting may be removed almost painlessly.

In regard to mortality, I am going to give the figures. I personally had fifty-two cholecystectomies with three deaths in the series, a mortality of 5.7%, twenty-six cholecystostomies with six deaths, or 23%, and eight choledocotomies with two deaths, or 25%. The figures for the cholecystostomies and choledochotomies seem high, but both might have been even cut in two, by not reporting cases complicated by acute pancreatitis, cases operated upon for acute cholecystitis early in the convalescence from operations for strangulated umbilical hernia and prostatectomy, and other cases in which operation was forced on us in the presence of serious handicaps. In these cases we have always taken the chance even if it was but a slight one. In the whole series we have only had one death in a case which seemed safe and simple, so that in favorable cases in young people the mortality ought not to be regarded or discussed as a serious matter.

DEATHS FROM CHOLECYSTECTOMY

1. C. D. Aug. 12, 1926. Age 49. Cholecystotomy and removal of stones fourteen years previous to cholecystectomy. Had dyspnea and palpitation, though no abnormality of heart was noted previous to operation. Anesthetic—ether. Operation difficult on account of adhesions. Stones and pus in gall bladder. Returned to bed in good condition. Rapid rise of pulse and temperature and death in two days. Death was ascribed to myocarditis, but infection might have played a part. Spinal anesthesia might have helped.

2. J. B. Sept. 13, 1926. Jaundiced. Very obese. Urine showed a trace of albumin and granular casts. Operation difficult. Would have done simple drainage except for a stone in the cystic duct which blocked it and could not be gotten up into the gall bladder. Given calcium lactate and digitalis before operation. Normal for two days after operation except for vomiting. Then pulse rose to 120 and grew weak. Probably a cardiac death. *Comment:* Incision of the cystic duct and removal of the stone without cholecystectomy might have helped.

3. W. U. Oct. 14, 1926. Age 31. General condition good. Symptoms for five or six years. Heart normal. Gall bladder contained stones and adhesions. Cholecystectomy and appendectomy without incident or difficulty. Normal convalescence 1st day. Second day temperature 102°, pulse 130. Fifth day distention, cyanosis, hiccough and vomiting. Consolidation lower base of right lung. Onset and progress thought to indicate embolism. Infection is of course a possible cause. This is the only death in the series in a simple, uncomplicated case with the patient in good condition.

CHOLECYSTOSTOMIES

1. A. O'G. April 27, 1927. Age 52. Symptoms for 16 years. Eleven years before cholecystostomy with removal of stones. Present illness fourteen months, attended with vomiting and chills. Obese. Anesthetic—Ether. Gall bladder bound in adhesions and difficult of access. Normal convalescence for five days. Died on eighth day in coma. Probably uremia. *Comment:* Spinal anesthesia might have been better in this case.

1. S. M. May 24, 1927. Age 43. Thin, dehydrated by vomiting for months. Constant abdominal pain. Neurasthenia. Had suffered from a pyelitis. Soft blowing murmurs at apex. Obstruction of duodenum was suspected, but bismuth series impossible on account of vomiting. Exploratory operation as a last resort. Gall bladder distended with thick bile, no stones. Drained. Died two days later with rapid rise of pulse and temperature. *Comment and question:* Was the thick, viscous bile which obstructed the duct and distended the gall bladder a result of the dehydration from vomiting?

3. M. G. Jan. 30, 1928. Age 54. One month before was operated upon by me for strangulated umbilical hernia, involving the small intestine and threatening gangrene. Went home in two weeks in good condition. Then began to have pain and vomiting and continued to suffer from these symptoms for two weeks at home. Returned to hospital emaciated and dehydrated, with a normal temperature, pulse of 90 and slight tenderness over gall bladder. Abdomen relaxed. Upper right rectus incision. On separating adhesions between the colon and the liver, a very large abscess was found containing thirty large gall stones. In its centre was a perforated gangrenous gall bladder. Removal of stones and drainage. Death the following day. *Comment:* If this patient had had her symptoms come on before leaving the hospital after the former operation and not waited two weeks at home with a perforated gall badder before calling for help, the result might have been different.

4. Mrs. J. P. Feb. 11, 1929. Epigastric pain for years. Jaundice one month. Obese. Edema of ankles. Heart sounds distant. Systolic murmur at apex. Seen in consultation by Drs. Denning and Curran, who stated that she was a poor operative risk on account of arteriosclerosis and myocarditis. She was digitalized and stimulated for one week. Pain and tenderness increased. Operated under spinal anesthesia. One large and two small stones removed and gall bladder drained. Consolidation of both lungs at bases soon appeared and death occurred on third day of broncho-pneumonia.

COMMON DUCT CASES

1. M. N. June 25, 1928. Age 65. Loss of weight in two years of fifty or sixty pounds. Vomiting, chills, chronic myocarditis. Arterosclerosis. Anesthesia: Local and gas with a little ether. Several stones removed from gall bladder and two from common duct. Coarse, muddy material washed out of common duct. Cholecystectomy and drainage of common duct. Died in ten days. Inanition. *Comment:* A very unfavorable case for operation.

2. B. H. Dec. 27, 1928. Acute attack within one week. Temperature 103° day of operation. Spasm and tenderness in epigastrium and slight jaundice. Gall bladder could not be found. Retroperitoneal mass in front of head of pancreas containing about four ounces of blood and pus. Opened and drained.

Single facetted stone found in common duct, which was explored up and down with the finger and no more stones found. Sound passed through papilla of Vater into duodenum. Tube in common duct and in pancreatic abscess brought out through a stab wound. Rise of temperature and death on third day. *Autopsy.* Two more facetted stones found in a pocket in the common duct close to the duodenum, where the finger had passed by them without detecting them. Gall bladder shrunk down to a small solid knob by old chronic inflammation. *Comment:* This case was complicated by pancreatitis. It is possible to overlook stones in a pocket off to one side of a dilated common duct in the presence of much inflammatory edema.

CHAIRMAN MILLER: Gentlemen, I am sure you will agree with me that this was a very interesting and a very instructive paper. My own thought as I listened to it was that the latter part of it was particularly interesting. The most complimentary thing I could say about it is that Dr. Lund was honest in the report of cases, as our dear old friend, Dr. John Monroe, used to be when he reported his cases—honest to the degree of leaning the other way.

I am sure this subject is one of very great interest to internists and surgeons alike. I have asked Dr. Royal P. Watkins, of Worcester, to open the discussion, and after his discussion, I hope there will be considerable other discussion or questions.

DISCUSSION

DR. ROYAL P. WATKINS, Worcester: In discussing a paper like this, there is not much to do except say amen and perhaps emphasize a few of the points.

We must, I believe, recognize that the only effective treatment for gall-bladder disease is surgical. The acute cases are usually easy to diagnose and demand prompt operation within the limits set forth by Dr. Lund. The chronic cases are often not easy to diagnose and many of them require very nice judgment as to the proper time to operate.

The diagnosis must be made chiefly on the history. The physical examination will help in a good many of the cases and the X-ray may help in some. The use of the Graham method has added greatly to the usefulness of the X-ray, but even with this it is too often disappointing. The evidence, both negative and positive, given by a G. I. series, I often find is as valuable as an X-ray of the gall-bladder itself. But even with the greatest of care, there is bound to be an occasional mistake in diagnosis.

All surgeons have probably had the humiliation of finding no lesion in the gall-bladder at operation, or of finding the cause of the symptoms in some organ other than the gall-bladder, or even of finding no lesion at all. I am heartily in accord with Dr. Lund's opinion of the man who removes a normal gall-bladder just because he started out to remove the gall-bladder.

It is not always easy, however, to tell by the look and feel of a gall-bladder whether or not it is normal. In a doubtful case of this kind, if careful exploration shows no other lesion to explain symptoms which have been sufficient to justify an operation, I believe it is justifiable to remove that gall-bladder.

As to the time to operate in the chronic cases; most, if not all, the complications of gall-bladder disease could have been avoided by early operation. In the early cases without serious adhesions, without systemic disturbances, without liver or pancreatic involvement, the operation is a simple one and should have practically no mortality.

A great many of the cases come to us after one or all of these conditions are present and it is this that makes gall-bladder surgery the serious thing that it is. In these late cases the help of the medical man is of tremendous value, and should most certainly be insisted upon, in just the ways set forth in Dr. Lund's paper. This help, however, is merely using the best means at hand to pull the patient out of a bad situation.

If the surgeon could get these patients before they get into that bad situation, nobody but the surgeon knows how much suffering it would save both the surgeon and the patient. Not all of these cases are seen early, or even first by the physician, but a good many of them are. I don't believe the average physician realizes what the delay incident to palliative and symptomatic treatment means to his patient. When he does and acts accordingly, he will be doing a much greater service than any medical consultant in the hospital can do in the late cases.

The time to operate on gall-bladder disease then is as soon as the diagnosis can be made, or as soon thereafter as the patient's condition will allow. (Applause.)

CHAIRMAN MILLER: The paper is now open for general discussion. I always feel as though a general discussion means a great interest in the presentation of the subject. I hope that there will be no holding back. I offered Dr. Jackson the chance to speak and he shook his head. We don't hear from Dr. Jackson very often these days.

If there is no further discussion, then we will take up the next paper. The next paper on the program is given by a man who is known to most of us for his medical research work on gastro-intestinal problems which have been presented before medical societies and in various publications. He has more recently been known to us through the publication of a most excellent and interesting book on diseases of the stomach and I have no doubt he is known to many of you as students, as associate in medicine at Jefferson Medical College. It gives me great pleasure to introduce Dr. Martin E. Rehfuss, of Philadelphia, who will announce his subject. I think that will be the better way. (Applause.)

DR. MARTIN E. REHFUSS, Philadelphia, Pa.: *Dr. Miller, Ladies and Gentlemen*—After the discussion we have had this morning and which I very greatly enjoyed listening to, perhaps I will have something very different to say. The subject that I am particularly interested in is the question regarding the actual work of the stomach itself. For more than sixteen years at Jefferson we have been studying the question of gastric digestion, and I think I am perfectly safe in saying that there is so much to learn about gastric digestion that we have merely begun to touch the province as to what actually goes on in the stomach. For more than six years we have studied the digestion of foods in the stomach, using more than 200 men and performing more than 1000 experiments, and in the normal stomach it is an amazing thing to note that the sequence of events which occurs is one which is perfectly beautiful to behold and one which is perfectly regular in its evolution. It is amazing to note that we are totally unconscious of what is going on after we eat a meal. It is equally amazing to note the extraordinary complexity of the human dietary and yet regardless of what we put into the stomach, the sequence of events occurs; the stomach gets down to work, the food is reduced to chyme and the whole question of digestive phenomena is begun and carried through to a conclusion.

Dr. Rehfuss presented his prepared paper. (Applause.)

THE MOTOR MECHANISM INVOLVING THE ANTRUM, PYLORUS AND DUODENUM*

BY MARTIN E. REHFUSS, M.D.†

THE clinician who has to do with gastrointestinal disease is constantly confronted with problems which involve this area of the digestive tract. It is probable that the sentinel of the entire digestive tract is the pylorus. Some observers call it the gateway, others speak of it as a valve, but all the evidence at our disposal would lead us to believe that the pylorus is a specialized portion of the stomach wall, vested with certain particular functions.

According to Alvarez, there is a fibrous tissue barrier at the pylorus, but the barrier is not complete, as some waves may run in both directions. The general conception regarding this part of the stomach is the belief that peristaltic waves pass on uninterruptedly to the pylorus and are followed by its opening. This is the conception of Cole, McClure, Reynolds, Schwartz, Wheelon and Thomas, and Kline; while those opposed to this conception are Case, Cannon, Groedel and Koester. My own belief is that this is true only regarding the height of digestion. Alvarez and myself believe that the pylorus shows definite discrimination in its action. Penzolda, Schule, Fermi, Hedbloom and Cannon found out that the pylorus separates the fine from the coarse particles. Kirschner and Mangold are of the opinion that there is an autonomous action on the part of the pylorus; while Phfugler and Ullman maintain that the tonus of the pylorus is affected both by acids and alkalies. There seems to be little doubt that pressure in the duodenum by food or liquid will hold back or delay gastric evacuation. On the other hand, if the material from the stomach is evacuated through a duodenal fistula, the stomach empties rapidly (Hirsh, Vonmering, Marvaix, Killing, Tobler, Cohnheim, Richel). Apparently this evacuation is the more rapid (according to Hirsh) the nearer the fistula is to the pylorus. Tobler and Carnot maintain that distension of the duodenum, with a balloon or single irritant, will delay gastric evacuation. Alvarez maintains that acid in the duodenum, as well as fat, will exercise a retarding influence on the stomach, but the acid response does not last as long. Marvaix and Best demonstrate that if acid is placed in the middle or lower third of the small bowel delayed gastric evacuation will occur. It is interesting to note that according to Openchowski the pylorus more than the rest of the tract will be inclined to respond with an increase of tonus to stimulation of the splanchnic nerves. Carnot and Cohnheim point out that excitement or fear closes the pylorus in dogs, while Wheelon and Thomas, as well as Karlsen and Carnot, claim that contraction of the pylorus can be produced by stimulation of almost any afferent nerve in the body. Perhaps no recent work on the pyloric function is so suggestive as that of these authors, and it is interesting to note that this mechanism, viz. the reflex effect of lesions outside the stomach can induce pyloric spasm. This is not at all inconsistent with the finding of increased pyloric tonus in many extra-gastric conditions.

Regarding the association of duodenal contractions with those seen in the pars pylorica, it is of interest to note that tonus waves in the duodenum are often dissociated with waves in the antrum and in the pylorus. This is pointed out by Joseph and Metzer, Wheelon and Thomas, Alvarez and Mahoney, Payne and Poulton, Ivy and Bloedman. The study of tonus contractions in the duodenum, particularly by the authors Ivy and Bloedman, is of very considerable interest. These authors, by means of a double gastric and duodenal balloon, point out that the

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duodenum during hunger manifests contractions synonymous with those that occur in the stomach, with the exception that frequently the duodenal contractions lag behind those of the stomach. The first part of the duodenum manifests more motility during hunger than the last part. They believe that the duodenal motility during hunger is dependent upon enteric reflex contractions within the stomach.

While they do not conclude that duodenal motility necessarily contributes to the sensation of hunger, they do cite and discuss evidence suggesting that nausea and headache of hunger is duodenal in origin. From a perusal of this work it is difficult to say just exactly where the gastric balloon really was in Ivy's experience, and it is significant that the duodenal balloon was not always in the first inch of the duodenum. They point out, as do other authors, that the passage of a contraction wave from the stomach over the pyloric sphincter to the duodenum, or, in other words, the existence of a relationship between gastric and duodenal motility during digestion, would seem to be apparent from the observations of Wheelon and Thomas. Luckhardt, Phillips and Coulson, studying the control of the pyloric sphincter, report that during digestion the peristaltic waves course toward the pyloric sphincter and on reaching it, relaxation occurs and the mass is hastily passed through the entire course of the duodenum. Cole, however, states that the duodenal cap is divided by a broad peristaltic wave, indicative of gastric peristalsis, which forces the chyme through the duodenum in finger-like masses. Ivy and Vloedman point out that their observations would indicate that with fluoroscopic examination in man in the majority of instances a contraction of the duodenal cap occurs, at least in the distal portion of the cap, after a brief pause following the arrival of the gastric peristaltic wave at the pyloric sphincter. Alvarez holds that the gastric waves stop at the pyloric sphincter, but grants that at least some influence passes over the sphincter to start the peristaltic rushes in the bowel. There is obviously a difference of opinion even among physiologists, as to the sequence of events which occur in this portion of the digestive tract.

Not only is there a difference of opinion regarding the sequence, and the question as to whether or not duodenal contractions follow rhythmically gastric peristalsis, but there is a very considerable difference of opinion regarding the motor phenomena of the antrum. Some observers are of the opinion that pyloric contraction waves proceed onward over the stomach wall, becoming progressively stronger and reaching the pylorus. Klein, in discussing this question, notes two types of peristalsis, one in which there is uninterrupted peristaltic waves such as we have just mentioned, and a second form in which the antrum contracts as a whole, and in which one might almost logically speak of this as an antrum systole. This is another problem of considerable interest, and one which is by no means solved.

A third problem of equal interest is the question of the pylorus as a factor in the regulation of gastric acidity. The Cannon theory of acid control, by which it is maintained that two-tenths per cent. hydrochloric acid on the gastric side opens the pylorus, is one which has been taught to every student of medicine. This question of acid control, however, is one which does not stand up to our modern knowledge of the motor phenomena of the pylorus. While it is probably true that acids on the pyloric side may tend to open the pylorus, it is equally true that concentrated high acids in the stomach probably slow up or inhibit gastric peristalsis. It is also true, as every clinician knows, that acid on the pyloric side is not necessary to gastric evacuation. A subacid as well as an anacid stomach will frequently empty even more rapidly, and there is also present a perfect sequence in rhythm, as demonstrated by X-ray study. There is much evidence against Cannon's theory; some of this is roentgenological, such as the evidence of Cole, McClure, Reynolds and Schwartz, in which it would seem that the opening of the pylorus is part of a sequential group of motor phenomena. The clinical evidence as shown by the behaviour of the stomach in the presence of achylia and with alkalis, demonstrates at least that acid on the gastric side is not necessary to open the pylorus; and the experimental evidence, with water and egg-white, suggests that such a theory does not explain all the phenomena connected with a normal stomach.

Oppenheim, in 1887, suggested that the control of the pylorus is vested as much in the bowel as in the stomach. In other words, the idea that pyloric function is not so much a function of the stomach as of the bowel, is not a new one. It is generally acknowledged by experimental observers that high acid in the duodenum delays gastric evacuation.

Another question of great importance within recent years, in which the pylorus is intimately associated, is the question of the regulation of gastric acidity. Since Boldyreff enunciated the idea that gastric acidity was regulated by the regurgitation of the alkaline duodenal secretion, this problem has received considerable thought. This observer pointed out that of all these substances coming in contact with the gastric acid, the most potent as a neutralizing agent was the pancreatic secretion. Saliva, pyloric secretion, succus entericus, bile—none of these were nearly as strong as was the alkaline pancreatic secretion. By instilling into the stomach of a dog, acids of concentrations higher than those of the normal optimum, he was able to show that these were reduced and evacuated into the duodenum in concentrations that were

acceptable to that organ. The discrepancy between the native gastric secretion which is now almost universally stated to be 0.4 or higher by authorities on this subject, while the optimum normal secretion found in the digestion of carbohydrates, for instance, is 0.15 0.2, suggests that there is some mechanism responsible for this extraordinary readjustment. The theory of Boldyreff, viz. that there was a regurgitation of the alkaline pancreatic juice, capable of inducing this phenomena, was one which was not without its attractiveness, because clinicians have observed a marked regurgitation with a total reducton of the secretion either to subacid or anacid figures. The thought, however, that this mechanism played a rôle in normal gastric digestion did not receive much attention until recent years. In 1919 we (Spencer, Rehfuss, Meyer and Hawk) attempted to investigate the problem in the normal human subject by studying the tryptic content. Spencer evolved a refined method by which trypsin could be determined in the gastric contents. By means of this method in the fasting stomach of healthy medical students, we were able to show that in most instances the tryptic content was inversely proportionate to the free acidity. Furthermore, we were able to demonstrate the presence of trypsin in increased concentration, when the acidity was reduced in the stomach. We furthermore found out that this might occur even in the absence of demonstrable bile. While these experiments suggested that alkaline regurgitation was probably to a certain degree a normal phenomenon, there was not sufficient evidence to prove that it occupied the principal rôle in acidity regulation. Shortly after this, Bolton and Goodhart in England, and Apperley in Australia, investigated this problem from another angle. They studied the neutral chlorides and pointed out that when the free acid dropped the neutral chlorides increased. This, they believed, was due to the regurgitation of an alkaline pancreatic secretion through the patent pylorus. Bolton then assumed that in all probability the hyperacidity was due to increased pyloric tonus which prevented the normal regurgitant mechanism from exerting its effect, with the result that a high acid curve ensued. These suggestions were all fascinating because they explained on the basis of pyloric hypertension and failure of regurgitation, the mechanism underlying gastric hyper-acidity. More recently, MacLean and Griffiths, in an interesting but, in our opinion not convincing series of experiments, suggests that the mechanism accounting for regulation of gastric acidity is not that which is due to regurgitation, but to an intra-gastric mechanism within the stomach itself. These authors maintain that the concentration of hydrochloric acid in the normal stomach is regulated automatically by the concentration of the gastric juice. The presence of a certain concentration of acid in the stomach, according to them, inhibits the secretion of acid, with the result that a neutral fluid, containing chloride, is secreted, which by dilution reduces the acidity of the gastric contents. Babkin, on the other hand, in an interesting communication before the American Gastro-Enterological Society, mentions the highly specific character of the different gastric cells and the probability that they are capable of manufacturing different types of juice. Not the least significant of these observations is the importance of a form of mucus which is secreted by a certain type of cell. Just what is the true explanation of the various phases of gastric digestion is not apparent at the present time, although strides are constantly being made in the right direction.

A little over a year ago, in view of the fact that Bolton and Goodhart had maintained the importance of pyloric hypertension, I constructed a gastro-pyloro-duodenal tube. By means of this it was possible to keep the pylorus patulous, and enable the contents of the duodenum and the antrum to mingle. At the same time it was possible to draw off specimens from the gastric antrum for examination. Using this method in proven cases of gastric hyperacidity Dr. Eads and I were able, in a fairly large number of cases, to cut the free acid curve in half, with the manifest evidence of duodenal regurgitation. It was not possible in all cases to accomplish this result, and in some cases, despite a patulous pylorus, gastric acidity remained as high as, or higher than before. This we could only explain on the assumption that the tonus of the gastric wall as a whole was greater than alkaline tonus, and prevented efficient regurgitation.

In view of the fact that it is impossible for us to know just how important is this alkaline regurgitant mechanism, it occurred to one of us to study it in a very different sort of way. With the aid of Dr. T. Earle Thomas, Professor of Physiology of the Jefferson Medical College, who has done pioneer work with the motor mechanism of the upper digestive tract, we succeeded in evolving a series of experiments for the purpose of demonstrating whether or not the sequence of events occurred in which duodenal tonus was probably greater than gastric tonus, and in consequence regurgitation might be facilitated. It was apparent from the literature that considerable study had been made regarding free balloons, but in very few instances was there any reference to an exact registration of events in a given segment of the antrum or duodenum. Ivy and Vloedman, to whom we have already referred, studied the duodenal hunger contractions, but there is nothing in their article to point out as to where the gastric balloon really was. The same criticism might be made of Templeton and Johnston's work: it is difficult to understand where the antral balloon

really was. We constructed a double tube containing two fractional tubes to which Dr. Thomas attached collodion balloons. By means of the metal tips within the balloons, it was not difficult to visualize the position of these balloons with a fluoroscope. By means of a recording kymograph, piston recorders, water manometers and the adequate employment of the tubes under fluoroscopic observations, we were enabled to get a series of graphs illustrating the motor phenomena of the antrum and duodenum. We were soon confronted with the difficulty of perfectly placing the antral balloon; in fact, we found it impossible to retain the antral balloon in place without anchoring it by previous inflation of the duodenal balloon. Furthermore, our double tube was so constructed that these two balloons were at a fixed interval apart, usually one inch or a little more, and so situated that the interval was a constant one. The simultaneous recording of the motor activity of the antrum and duodenum in the digesting stomach of a normal, untraumatized human being, such as we tried in vain to find in the literature, we found to be difficult owing to the fact that both balloons either passed into the duodenum, or were withdrawn into the fundus of the stomach. We did succeed, however, in getting a small series of observations actually showing the sequence of events during digestion. It is interesting to note that the duodenum is much more irritable than the stomach, and, as Ivy pointed out, there is undoubtedly evidence to show marked motor activity in the first part of the duodenum in the fasting stomach. We encountered series of sequential contractions similar to peristaltic waves, both synchronous with and following gastric hunger contractions. We were able to demonstrate that the mean low pressure in the duodenum at times was perceptibly higher than in the gastric antrum, and, therefore, conditions were conducive to regurgitation. We were also able to demonstrate that there were periods of increased irritability and tonus of the duodenum even when the antrum was relatively quiet, again suggesting that a condition conducive to regurgitation was present. It is true that this method is open to all the criticism that the balloon method of recording entails, and that some of the motor phenomena of the duodenum may have been induced by the balloon. The fact, however, that the sequence of events was such would suggest at least that in the fasting stomach at intervals a similar, if less marked, series of events probably occurs. In our studies we noted simultaneous tracings of the first and second part of the duodenum during digestion, and the similarity in the response, and the absolute rhythmicity suggesting that these waves were part of general peristaltic movement which was sequential in character. The study of two balloons in the antrum, however, leads to considerable speculation. Examination of simultaneous points would lead the observer to believe that perhaps the antrum may contract as a whole. There is certainly considerable work in this particular field, and our studies would suggest that perhaps both methods of peristalsis are possible. Among the interesting observations which came to our attention was the reaction of the fasting stomach to food. In our graphic charts it is apparent that after the introduction of food the stomach gradually builds up tonus. This is very pronounced in some of the records, but it is of interest to note that simultaneously with the building up of tonus on the gastric side, there is an increase in gastric acidity.

A careful study of the curves at our disposal which show very well antral peristalsis, and, in many instances, duodenal peristalsis, would suggest that duodenal peristalsis may occur at the down stroke or even the relaxation period of the antrum. It is not difficult in studying these cases to realize that a sequence of events can occur during one phase of gastric peristalsis at which some regurgitation might occur. While our results are necessarily limited, there are evidences in some of the graphs to suggest that this is possible. Not only does this seem apparent from some of our studies on men, but a study of the graphs of Wheelon and Thomas, particularly the reconstructed graph, suggests that the top of the duodenal stroke is probably at the termination of the pyloric contraction. It may be argued that our experiments do not take into account the tonus of the pylorus, but the very complete and convincing studies of Wheelon and Thomas suggest that pyloric tonus is part and parcel of the antrum contraction, synchronous with it and also following it. I believe their evidence is sufficient to justify this belief, and I can see no evidence that we are acquainted with on the human subject which would deny this assumption. Certainly the pyloric contraction as seen on the x-ray screen, or at least the pyloric wave, follows immediately after antral contraction and is practically simultaneous with it. This is the conclusion of these observers on dogs, and seems to be borne out in either way by what we know of human beings. The behavior of the first part of the duodenum is not so clear, but there is at least evidence to make us believe that intervals occur which are conducive of regurgitation. In one experiment which we attempted to perform with an antral balloon, and also a fractional tube, in order that we could record antral contractions and examine the secretion at the same time, which was a failure, the instrument was so constructed that a balanced heavy tip was supposed to maintain the antral balloon in place. In every instance the balloon receded back into the cavity of the stomach, showing the power of the antrum to accomplish this effect. We found that the only way to anchor the antral balloon

was to inflate the balloon in the duodenum which was attached at a fixed point to an antral balloon. We feel, therefore, that these experiments in gastric and other intestinal motor functions, lend color to the belief that at certain intervals, in the fasting stomach, and again at a very brief period during the active peristaltic cycle, are conducive to duodenal regurgitation. We hope in the future to present further evidence regarding this mechanism in the digestive tract.

CONCLUSIONS: 1. Evidence is presented to suggest that the sequence of events in the antrum, pylorus and duodenum is such in the fasting stomach, and at certain stages in the digesting stomach, as to permit or at least be conducive to duodenal regurgitation.

CHAIRMAN MILLER: It is a platitude to say we have listened to a most excellent exposition of the subject. I hope that there are a great many questions to be asked, perhaps some additions. The discussion will be opened by Dr. Edward S. Emery, Jr., of Brookline.

DISCUSSION

DR. EDWARD S. EMERY, JR., Brookline: *Mr. Chairman, Ladies and Gentlemen*—Dr. Rehfuss has given us a very pretty demonstration of some of the work that is to be done in studying this phase of medicine. He has emphasized the fact that it is a very difficult problem, but I would like to emphasize that even more. The pylorus has been looked upon in times past as the gateway from the stomach to the intestines, rather than the other way around, and because of its position, has always attracted a great deal of attention, with the result that a great deal of work has been done upon it. One of the earliest ideas regarding the control of the pylorus was the acid one, or chemical regulation, first brought out by Hurst and Von Mehring; Cannon felt that the acid was the important factor. Later work has brought that into question. This later work, such as McClure and Reynolds did in Boston, of injecting acid directly into the duodenum and failing to observe a contraction of the pylorus by the fluoroscope; the well-known observations that the pylorus will reject large pieces of meat, and yet will let large foreign bodies such as safety pins go through; the work on the nervous control of the pylorus, such as done by Carlson and others, where they find the vagus in most cases causes contraction of the pylorus but in a certain number of animals it also causes relaxation, whereas in a certain proportion of animals stimulation of the sympathetic caused relaxation, but in a certain number caused contraction; all these are findings that haven't been explained satisfactorily as yet.

Then there is other work such as Cole has done with the X-ray, suggesting that spasm of the pylorus occurs as a reflex disturbance from the ileocecal region, and from other places, such as Dr. Rehfuss has spoken of. It has always seemed to me that until we get the final evidence, we might as well assume that all these things may occur under normal conditions. It is perfectly conceivable that we can have a muscular coördination perhaps to a degree. We can have a certain chemical regulation and at the same time we can have a very distinct nervous regulation. Under abnormal circumstances, one of these may offset the others, that is, if we have a reflex action, we can get spasm as a result, and that may be sufficient to overcome any chemical regulation that may occur.

From a practical standpoint, of course, what we would like to know, if the patient comes in complaining of symptoms, is this: Is the pylorus acting normally, or is it not? If it is abnormal, what is the effect of that abnormality, and what is the cause of the abnormality? In other words, are we dealing with an abnormality in chemical regulation or in nervous regulation or in muscular coördination.

Finally, of course, a very practical point which we can't answer perhaps at the present time is, what are we going to do if the pylorus is acting abnormally? It is a very difficult problem, and we don't know the answer yet. We know so little about the gastro-intestinal tract. We don't know, for example—or put it another way, we find that the acidity as we ordinarily do it ourselves, seems to vary considerably in normal persons, if by normal persons we mean those who do not complain of gastro-intestinal symptoms. We know that in cases of achylia gastrica, so-called, where there is no free acid secreted, we oftentimes get diarrhea, but not always. Approximately twenty-five per cent. of the patients with an achylia gastrica show diarrhea. On the other hand, about fifty per cent. may complain of constipation. So that we don't know as yet what the distinct normal and abnormal is. As time goes on, we have got to know these things before we can come to a prac-

tical solution of our problems. In the meantime the work, such as Dr. Rehfuss is doing, will be the means of opening that problem to us. He is the man who has done more in the study of gastric disease by means of the stomach tube than anybody else, and I consider it a great privilege that we have been able to listen to his discussion. (Applause.)

Dr. John Homans, Roxbury: I am very much fascinated by this subject, even though I am a surgeon. Some years ago I indulged myself in some experiments upon the motility of the stomach, using the old-fashioned system of the large bag in the fundus so that I am a little bit familiar with this method of doing things. I think that it would be very interesting if Dr. Rehfuss would explain a little more fully to this audience, who. I assume, are quite as ignorant about the matter as I am, the difference between the hunger contractions and the contractions which go with digestion itself. In my experience, the hunger contractions are very peculiar things and I can't quite see how conclusions can be drawn as to the behavior of the stomach, the antrum and the beginning of the duodenum, from the hunger contractions alone, because when hunger contractions are going on, all other activity ceases except perhaps tonus. When digestion begins, the hunger contractions stop. And apparently in pathological conditions the hunger contractions cease while the patient is having pain, but are resumed as soon as the patient becomes comfortable. There are a great many other fascinating things of a similar sort which I am sure Dr. Rehfuss knows more about than I do. If what Dr. Rehfuss says in explanation of the acidity of the stomach in relation to its muscular tone is true (and I should suppose it was true, for certainly his experiments are very striking), it makes clear why the patient with a duodenal ulcer has an increasing acidity and it explains why our treatment at least produces a pleasant effect upon the patient, whatever it does to the ulcer. If what he said in respect to the behavior of the duodenum and antrum having a certain reciprocal muscular activity is true, then we have to think of the pylorus as a place where the food can be passed back and forth quite readily, which I am not quite willing to accept. It would appear from what he says that at one time the tone in the duodenum is higher and food goes back into the stomach; at another time the tone in the stomach is higher and material goes into the duodenum. It would even seem as though there were a to and fro motion through the pylorus. I don't believe he quite meant to leave that impression. Of course, we think of the stomach as being a churning organ itself, and that the pylorus merely opens and closes to let the food out. However, his explanation of the return of duodenal contents to diminish a high acidity might perfectly well be regulated in such a way as he suggested, as an occasional phenomenon and not, I think, as his curve suggests, as a constant phenomenon.

Chairman Miller: I very much dislike to cut off such an eloquent flow of language, but the other end of the entertainment, the Councilors, are notifying me constantly it is time to let the men who are Councilors leave here. Personally, I am much more interested in what we have here, so I am going to ask anyone who cares to ask questions or to discuss this paper further to do so without regard to the Council meeting.

If there is no one else, I would like to ask Dr. Rehfuss if he has something to say.

Closing Discussion

Dr. Martin E. Rehfuss, Philadelphia, Pa.: I will say it in a very few words.

So far as that is concerned, regarding the churning, it sounds like a perfectly logical thing. You are all capable of drawing your own interpretation of these things. I have shown you mine. My idea is that there is a sequence forward; in other words, the duodenal sequence is forward, not backward, but I mean it is possible that owing to the increased tonus of that point and relaxation on the other, a small amount of material gets in.

Regarding hunger contractions, I would never start an argument like that, because a great many people believe that hunger contractions are exaggerated peristaltic contractions. In any event, it doesn't make any difference what they are; they are simply contractions which mean increased pressure. That is the only point I would like to get across.

Finally, the only thing I have to say regarding the question of what all these things mean, I don't know. I was simply looking to see whether any period was conducive to this thing which we found out; namely, there is occasionally a regurgitation of alkaline material in the stomach. It occurs very much more in the fasting stomach and the acidity of the fasting

stomach is one-half of the digesting stomach. Then it occurs during the early phases of digestion and probably never occurs at all during the activity. At any event, I am sure, your interpretation and my own belief is that there is a period during which a small amount of alkaline material can get in the stomach. It is needless to point out the whole tendency is downward all the time. In other words, it goes downward and the food goes on.

I would like answers to a lot of questions myself because there are a good many I can't answer.

I am asked if the regurgitation of bile is a pathological phenomena. In 100 normal students, we found that fifty-five per cent. had macroscopic bile in their fasting stomach contents. The explanation of that is it is not only duodenal regurgitation, but bile regurgitation. In other words, it is very probable the pylorus is relaxed during the fasting of the stomach and you can see that great group of contractions occur and that there will be a certain amount of biliary reflex. I don't believe that means anything.

VOICE: What would vomiting bile mean?

DR. REHFUSS: I believe vomiting bile is reversed peristalsis. I don't see what other explanation you can have. If you have obstruction below the common bile duct, you not only have vomiting bile, but you have bile all through the whole gastric phase. The only thing that is very interesting about all this is that even with an alkaline stomach, as the Doctor will tell you, the stomach empties the same. The acid mechanism very probably explains certain features of the problem, but an achylia stomach acts exactly like a hyperacid stomach, and more rapidly.

I screened 20,000 stomachs. I have kept count of them, and I know for a fact a stomach that has no free acid shows its peristaltic waves and empties in a normal way, exactly like a stomach that has acid. On the other hand, that doesn't explain the normal phenomena. There is a great deal we have to learn and a lot I have to find out.

My explanation is if there is a low grade gastro-intestinal catarrh, then achylia gastrica will be followed probably by a looseness of the bowels. You will find out a lot of them are like that. On the other hand, there is the type, pernicious anemia perhaps, where there is a disappearance of acid and there may be actual constipation. I think there is no one explanation for those things. The classical case is not one hundred per cent.; it is just as the Doctor said, the classical case of achylia gastrica produces constipation. Yet when I was in France and Berlin, I thought every case was that way; I know that isn't so.

CHAIRMAN MILLER: Has anyone else a question to ask Dr. Rehfuss? If not, the period of our exercises has elapsed and a motion to adjourn will be in order.

It was regularly moved, seconded and carried that the meeting adjourn. The meeting adjourned at twelve-ten o'clock.

The following officers were elected to serve for the ensuing year in the Section of Medicine:

Chairman—Dr. Francis W. Palfrey, Boston.

Secretary—Dr. Albert A. Hornor, Boston.

VERMONT STATE MEDICAL SOCIETY

SOME OBSERVATIONS ON SPINAL ANESTHESIA*

BY GEORGE R. ANDERSON, M.D.†

I HAVE selected the subject of spinal anaesthesia, or more properly speaking spinal analgesia, in the hope that it will be of interest to the majority of the physicians as well as the surgeons at this meeting. Anaesthesia is of equal importance to the patient, operator, and attending physician. How many times do we hear the patient remark "I would not dread an operation were it not for the ether." Since the days of Hippocrates members of our profession have diligently sought the ideal anaesthetic. It is my candid opinion that it has not yet been discovered. It is true great strides have been made since the introduction, eighty years ago, of ether, which came as a great boon to suffering humanity. Anaesthesia by means of general anaesthetics has become less to be dreaded as advances in technique, employment of gas oxygen ether sequence, and still more recently, ethylene. Anaesthesia with anoci-association, as developed by Crile, several years ago, although never popularized to any great extent, made for great improvement over then existing methods of general anaesthesia. The more recent investigations and employment of sodium amytal, intravenously, undoubtedly marks a revolutionary step in the production of general anaesthesia.

At this time no topic is more frequently written about and scientifically discussed than local, spinal, lumbar, and sacral anaesthesia. All of these methods had their inception in the discovery of cocaine about 80 years ago, but it was not until 40 years ago that Corning, an English physician, used it in the spinal canal, experimentally, on dogs. However, it was not until Bier began his investigations, many years later, that spinal anaesthesia became an established surgical procedure. The first human subject to receive spinal anaesthesia was Bier himself. He became very enthusiastic at the result of his experience.

After his experiment the method was adopted by surgeons throughout the world, especially in America and France. I believe the first operation performed in this country is credited to Tate. He did an osteotomy of the leg in 1899. The method fell into disrepute because many men employed it without having proper knowledge of its effect on the animal body and the technique of application. Fortunately, it was persisted in by a few who sought safer methods and drugs, for up to this time only cocaine had been used. With the discovery of stovain and novocain in 1905 spinal anaesthesia was attended with more favorable results, as both of these drugs are much less toxic than the alkaloid cocain.

Babcock, to whom great credit must be given, has produced spinal anaesthesia in more than 15 thousand cases, with extremely low morbidity and mortality. Labat, the chief French exponent, has administered novocain in many thousands of cases at the Mayo clinic and post graduate hospital in New York, with very excellent results. However it was not until Pitkin presented his epochal paper at the meeting of the college of surgeons a year ago, that spinal anaesthesia received country wide attention and was enthusiastically adopted in many hospitals and clinics. Following his presentation of controllable anaesthesia by means of heavy and light solutions, his hospital in Teaneck, N. J., became the mecca of thousands of interested anaesthetists and surgeons.

The physiological reactions due to spinal anaesthesia are interesting. Novocain in solution in the spinal canal is rapidly absorbed by the spinal nerves blocking the passage of sensory and motor stimuli. As it passes upward in the canal to a level necessary for the performance of abdominal surgery, most of the spinal nerves supplying moter stimulation to the sympathetics are paralyzed, causing vasodilatation, with resulting fall in blood pressure. The extent of this fall in blood pressure depends on the amount of sympathetic paralysis. This feature, one of the most alarming to the uninitiated, is controlled, in part, by the Trendelenberg position, plus the administration of ephredine or adrenalin in the Pitkin technique. It causes the blood to gravitate to the brain, thereby preventing cerebral anemia. In our experience a combination of both of these methods has prevented symptoms of great vascular relaxation or cerebral anemia. The sensory conduction is lost before the motor function is affected. In addition to the action of the drug on the vasomotors, blood pressure is also lowered by paralysis of the abdominal muscles, with resulting relaxation. The effect on the intra-abdominal organs is truly remarkable and most gratifying. The intestines are contracted, often to the size of one's finger. The stomach and hollow viscera are almost completely devoid of gas. The intestines slide into the upper abdomen, with the patient in the Trendelenberg position, without the use of packs, and the entire picture has been described by Russell as a "Surgeon's Paradise." In high operations, patients will occasionally complain of difficulty in breathing, which indicates partial paralysis of the chest wall. This quickly yields to a few minutes of oxygen inhalation. The effect on the respiration and circulation is dependent al-

*Read at the annual meeting at Bellows Falls, October 10, 1929.

†Anderson—Surgeon-in-Chief, Brattleboro Memorial Hospital. For record and address of author see "This Week's Issue," page 1116.

most entirely upon the height of the anaesthesia, and is caused by paralysis of the sympathetic accelerators. Pallor is almost always present and indicates that the anaesthetic effect has been obtained.

Pitkin and Hooper began their efforts to discover an ideal anaesthetic in 1927. The goal was to obtain a solution which would be certain in effect, constant in duration and controllable, without the dangers associated with methods then in use. They worked out a solution, lighter than the spinal fluid, giving it the name spinocain. The specific gravity of spinal fluid is 1.007. The specific gravity of spinocain of 1.0005. Spinocain is novocain, alcohol, strychnine, starch and normal salt solution, and because of its lighter specific gravity when injected in the spinal canal, rises rapidly to the highest point, as the air bubble in a carpenter's level when the end is elevated. Leaving out the alcohol and adding propantriol, they obtained a solution with a specific gravity of 1109, much heavier than spinal fluid, which rapidly gravitates to the most dependent parts of the dura. With the perfection of these two solutions, the problem of controllable anaesthesia became greatly simplified. There still, however, had to be devised a method of controlling the vaso-motor constrictors. Strychnine, combined with the novocain, gives results, but is not adequate in safe doses to entirely counteract the action of the novocain, therefore ephedrine was used. This, as you all know, is the active principal derived from Ma Huang, a Chinese herb, which has been known and used in Chinese medicine for more than 5000 years. It is closely related to adrenalin. In its pharmacology it is a powerful stimulant to the sympathetic nervous system and raises blood pressure in this way. Pitkin combines it with novocain, using it as a preliminary injection in the skin and the soft parts between the spinous processes. While ephedrine has the peculiar blood pressure raising properties when administered before spinocain is used, it seems to have little, if any, effect when used any time within two hours after the administration of spinocain. This is quite contrary to the action of adrenalin, which, in the presence of spinocain, quickly produces an increase in the blood pressure, whether introduced intramuscularly or intravenously.

The technique of using spinocain varies considerably from ordinary spinal anaesthesia as employed by Babcock and Labat. It is the only one with which we are familiar. I think one using spinal anaesthesia should thoroughly familiarize himself with the method of his choice, and visit the clinics where it is being used.

First a word as to the preliminary preparation of the patient for spinal anaesthesia. I believe this should differ in no way from the accepted preoperative preparation where general anaesthesia is contemplated. The blood pressure reading should be carefully taken and noted on the pressure recording card sent with the patient to the operating room. Where possible we clean the bowels out thoroughly, otherwise the relaxation of the sphincters and the increase in intestinal peristalsis may end in embarrassing results on the operating table. We thoroughly alkalinize the patient, and in dehydrated cases, administer fluids orally, subcutaneously, intravenously and by rectum, when time and circumstances permit. It is our custom to administer a hypnotic to insure a good night's sleep. In unusually neurotic types, we follow Crile's suggestion of anoci association and administer a small dose of morphine or morphine and hyoeine, or ½ an h. m. c. tablet in the early morning. This may be repeated in an hour in whole, or in part. We like to have them sleepy when they arrive in the operating room. In very young or very old subjects this preliminary treatment is often omitted. We prefer to have the eyes of the patient covered with small sponges strapped over each eye with adhesive, and in certain cases, the ears plugged with absorbent cotton. This, however, is not a hard and fast rule, as some cases should be psycho-anaesthetized, and here is where one of the most important phases of spinal anaesthesia comes in. Fortunately we have an excellent anaesthetist, who has the happy faculty of psycho-anaesthetizing her cases, keeping them interested in subjects other than their impending operation. We have installed in our operating room a pair of ear phones, which are connected up with the regular radio set in the wards and private rooms, and in some cases, this is a valuable adjunct; first, becauses it keeps the patient's mind occupied with pleasant thoughts, and secondly, prevents him from hearing conversation pertaining to findings, etc., which he should not know. It also prevents him from hearing the necessary clatter of instruments and basins. We try to have everything in readiness when the patient reaches the operating room. The simple instruments required for this procedure are placed on a small stand at the right and within easy reach of the operator. The same aseptic technique is carried out as in an abdominal operation. The armamentarium consists of two syringes, a 2 c.c. and a 4 c.c., a sharp pointed bistoury and two needles with stilettes in place, size 20 or 22 gauge. These should be made of rustless steel, as they retain their sharp point longer, less apt to break and will bend readily. The needles and syringes supplied by the Becton and Dickinson Co., with the Luer-Lock feature, have been most satisfactory in our hands. A small syringe with its fine needle, contains the ephedrine-novocain solution for injection of the soft parts and the skin. The larger needle has the short bevelled end, which cuts a small trap door, where it passes through the dura, which opening closes by intradural pressure, when the needle is withdrawn. This prevents the seepage of the spinal fluid, which so often

results in headache and backache. This small needle minimizes trauma and is much to be preferred to the old style needles used in spinal taps. The ampules containing the spinocain and ephedrine solution, should be carefully marked and sterilized by immersing in 70% alcohol. Great care must be exercised to distinguish between these ampules. The contents of the ephedrine ampule, 1 c.c., should be drawn into the small syringe and the spinocain into the larger.

The patient is then placed upon the operating table upon his side. The psycho-anaesthetist takes her position by the side of the patient, facing his abdomen. With one arm about his neck and the other under his knees, the chin and lower extremities are approximated as closely as possible. Hips and shoulders should always be in a vertical position to avoid twisting of the spine. This results in the backward curving of the spine and separates the spinal processes to their widest extent. The skin of the back is then sterilized with a 5% tincture of iodine solution, extending from the crest of the ilium and the sacrum up to the middle of the dorsal vertebrae. The fourth lumbar interspace is located, as in performing an ordinary lumbar puncture. This space is selected when anaesthesia is desired in the lower extremity or perineum. In operations in the upper abdomen, quicker results will be obtained if injection is made through the second or third lumbar interspace.

The paramount thought and endeavor should be, not to hurt the patient at any time, because he may lose faith and confidence in the method, which would be hard to regain and prove to be the direct cause of a non-successful anaesthesia. Pitkin says to be successful with spinal anaesthesia, the technique should be developed to such a finesse that the patient is never subjected to pain, and is carried through the operation in a happy and cheerful frame of mind.

The foot of the table is elevated so that the patient is in a 5 degree Trendelenberg position. This is estimated by the Pitkin Tiltometer, which is attached to the operating table. Spinocain should never be injected with the patient in any position except the above. The fourth lumbar interspace is determined in the usual manner, thumb and finger is then passed up the spine to the first lumbar vertebra, and the widest interspace found, may be selected for the point of puncture. Grasp the spinous process above the interspace selected between thumb and forefinger of the left hand, make the skin taut by pressure toward the abdomen, and insert the small sharp needle attached to the ephedrine-novocain solution syringe. Very little or no discomfort is caused by this procedure. The needle is inserted for its full length, forcing the fluid in advance. We have found that a wait of two or three minutes following this injection, permits the absorption of the ephedrine and its effect upon the vaso-dilators is more satisfactory than when the spinocain is injected immediately into the dural sac. In other words, it anticipates the action of the novocain on the vaso-dilators, and in a large measure, prevents their action. The small wheal induced by the ephedrine-novocain solution, is punctured by the sharp scalpel, and a small rather blunt spinal needle is inserted through this aperture, passing through the supraspinous and intraspinous ligaments, directly in midline and at right angles with the surface of the skin. The needle should be passed directly in the middle of the interspinous space. When it reaches the dura, in the majority of cases, a distinct snap will be heard followed by the loss of resistance previously felt. The stilet of the needle is then removed and, if the needle is within the dura and the arachnoid, spinal fluid will immediately flow. If it does not, rotation of the needle will often cause it to do so. If no spinal fluid appears and bony resistance is felt, your needle has undoubtedly been deviated and inserted too deeply, striking the body of the vertebrae. In this event, it should be withdrawn to the skin and reinserted at a slightly different angle. Occasionally, the first drop or two of spinal fluid will contain blood, this usually ceases after two or three drops, and the syringe containing the spinocain may now be attached. The plunger should be withdrawn and should be readily followed by the spinal fluid, if the point is within the dural sac. Never inject spinocain unless this can be demonstrated, but disconnect your syringe and reinsert, or withdraw the needle very slightly until the spinal fluid flows freely into the syringe. Of course unless the spinocain is injected within the arachnoid space, anaesthesia cannot occur.

When anaesthesia is desired in the perineum only, 1 c.c. of the spinocain should be administered, preferably in the fourth lumbar interspace, and the patient immediately placed in 15 degrees Trendelenberg position. If anaesthesia is desired in the lower extremities, 2 c.c. of spinocain should be used, as follows: aspirate 1 c.c. of the spinal fluid into the syringe containing the spinocain and inject ½ of its contents, then again aspirate 1 c.c. of spinal fluid into the syringe and inject the full amount. This amount of anaesthetic solution is sufficient to bathe all of the lumbar nerves. The table is then placed in 10 degrees Trendelenberg position. For anaesthesia to the umbilicus, we have found it best to use 3 c.c. of spinocain, as follows: into a 4 c.c. syringe, aspirate 1 c.c. of the spinal fluid and inject ½ of its contents, then aspirate 2 c.c. of spinal fluid in the syringe and inject the contents of the syringe, placing the patient in a 5 to 7 degree position. If you wish the anaesthetic to reach the 6th or 7th dorsal vertebra, with anaesthesia to the costal margin, draw up 3 c.c. of spinocain into a 4 c.c. syringe, aspirate 1 c.c. of the spinal fluid, inject ½ of its contents, again aspirate 2 c.c. of the spinal fluid and inject ½ of its contents and repeat once

again to expand to a total of 8 c.c. into the dural sac, then immediately place the patient in a 3 to 5 degree Trendelenberg position. This technique, as worked out by Pitkin, works very satisfactorily in normal individuals. If we are dealing with a very heavy patient or of a patient of six feet, with a long spinal canal, we have found it necessary, to obtain satisfactory results, to increase the dosage of spinocain to 3½ c.c. Sise has used 4 c.c., with satisfactory results. When the heavy solution, with which we have had no experience, is used, the patient should never assume the Trendelenberg position, but should receive his spinal tap while sitting up on the edge of the table with the back bowed outward and elbows resting on the knees, the head bent forward with the chin on the chest. The 4th lumbar interspace is located in the usual manner and the procedure carried on as described in the lateral position, except that one drop of the spinal fluid should be allowed to run from the needle to reassure yourself that it is in position and ½ c.c. of the heavy solution should then be injected. The patient should remain seated for three or four minutes to allow the anaesthetic to become fixed, then placed in a Fowler's position of 15 degrees. To secure anaesthesia of the legs, aspirate 2½ c.c. of the spinal fluid into the syringe containing ½ c.c. of the heavy solution and inject the contents. Immediately place the patient in 12 degrees Fowler's position. The heavy solution is not adaptable for operations in the pelvis or lower abdomen, unless the patient suffers from asthma or some cardiac condition and cannot breath in a recumbent position. Should occasion require, anaesthesia may be induced by placing .75 c.c. of heavy solution in a 4 c.c. syringe and with the patient on his side in 8 degrees Fowler's position, 3 c.c. of spinal fluid is aspirated, injecting ½ of the contents of the syringe, again aspirate 2 c.c. of spinal fluid and inject the full contents. Upon withdrawing the needle, it is well to place a drop of collodion over the opening and wash off the iodine with alcohol Turn the patient on his back and adjust the table to the degree of Trendelenberg or Fowler's whichever is indicated. The solution should be injected into the spinal canal at the rate usually employed in giving a hypodermic injection subcutaneously. The line of anaesthesia can be readily determined by familiarity with the technique, by the accuracy of the extent of the expansion, and by determining the degree of Trendelenberg, or Fowler's position, if the heavy solution is used. If for any reason the aspiration and mixing cannot be accomplished satisfactorily, the head of the table may be raised until anaesthesia has reached the desired point, meanwhile, watching the patient very closely, as the fluid extends more rapidly up the spinal canal than does anaesthesia.

Immediately upon the arrival of the patient in the operating room, blood pressure reading is taken and noted on the card provided for this purpose. A second reading is made immediately after the injection of spinocain and thereafter every five minutes during the operation. Readings are also made every fifteen minutes for two hours after the patient returns to his room. We also take the precaution of maintaining a slight Trendelenberg posture while the patient is being transported from the operating room and for two hours after the injection, after he has returned to his room.

In spite of every effort, it is a fact that occasionally spinal anaesthesia will be a complete failure. This is almost invariably due to faulty technique, either improper injection, too small a dose, or the wrong degree of Trendelenberg. It is remarkable how easily the spinal needle can be withdrawn from the subdural space while attaching the syringe, therefore the necessity for always drawing some of the spinal fluid into the barrel of the syringe to make sure that you are within the canal. The so-called "dry tap" is also due to faulty technique, the needle passing outside the dural canal.

I have failed to enter the dural sac in two cases. The first was a patient with an extremely rigid spine with a very narrow interspace. The second was in a very large patient with an abnormally fat back, the needle not being sufficiently long to reach the canal. I have failed in two cases to obtain satisfactory anaesthesia; the first due to too small a dose, with improper expansion, which I completed easily under novocain locally injected, and a second, a case of general peritonitis following volvulus of the small intestine, in a man 6 ft. 4 in. In this case I failed to allow for an abnormally long spine, in which I used slightly less than 3 c.c. of spinocain, expanded to 8 c.c.; anaesthesia was not wholly satisfactory and we found it necessary to resort to gas oxygen sequence.

As a rule anaesthesia occurs from five to ten minutes after injection within the spinal canal. The patient is usually ready for the incision by the time he has turned on his back, and the operative field prepared. The duration depends on the size of the dose, the minimum amount specified lasting thirty minutes, and the maximum, about two hours. We have not found it necessary to piece out with general anaesthetic.

Now a word as to the postoperative phases. The almost total absence of postoperative nausea and vomiting is noted. Those cases in which it occurs I believe are susceptible to narcotics. This is quite the contrary in inhalation anaesthesia. There is a marked difference in morbidity. No shock, abdominal distention, postoperative sweating, restlessness nor intolerable thirst, with parched mouth, to my mind one of the most disagreeable complications following general anaesthesia. I have yet to see a case of acute dilatation of the stomach, nor have I found it necessary to do a gastric lavage in

spinal anaesthesia cases. Of course postoperative pneumonia, pleurisy, acute bronchitis and other pulmonary complications, so frequently seen after ether induction are absent, all of which makes for a much easier and happier convalescence. What can be more agonizing than coughing or retching and vomiting, often lasting two or three days, with which we are so familiar, following long and complicated abdominal operations. Ileus, practically never occurs.

One cannot truthfully say that spinal anaesthesia is without its complications and fatalities. Of the former, the most unpleasant are the persistent headaches, backaches, palsies and neuroses, all of which are directly due to faulty technique. Paralysis of respiration and of circulation are due to too high anaesthesia, affecting the cervical nerves leading to the diaphragm. This condition is readily relieved by artificial respiration and administration of oxygen and carbon dioxide. I can find no record in literature of poisoning from novocain administered intradurally.

Sise has recently reported in the NEW ENGLAND JOURNAL OF MEDICINE,* a mortality of one in a hundred, in Greater Boston. Every new method is tried out by any number of incompetent or unprepared operators. Undoubtedly many of these cases are moribund and would not have survived under any type of anaesthetic. The mortality in the Lahey Clinic in June was about one in a thousand.

We have not used spinal anaesthesia in any operations above the diaphragm, but have performed nearly all of those below the diaphragm with gratifying results, and much satisfaction to ourselves and the patient. It is an ideal anaesthetic in prostatectomies. A most interesting illustration was a one stage operation on a man in his ninetieth year, who came through with no complications, very little discomfort and very happy end results. In pelvic tumors, ruptured ectopics, and caesarian sections, it seems to be the method par excellence. To those who have never performed a hemorrhoidectomy under spinal, a revelation awaits.

My experience and results with the Pitkin method have been extremely satisfactory. In my whole series of some under a hundred cases, I have not observed any alarming symptoms or deleterious effects. I have lost one case, a prostatectomy, a very poor surgical risk, who died two months following the operation. I am sure this was not influenced in any way by the anaesthetic.

CONCLUSIONS

I believe that spinal anaesthesia or analgesia, when skillfully administered, with proper technique, gives complete absence of pain, complete relaxation, with resulting ease and rapidity of operation.

Convalescence is associated with far fewer complications and definitely lowered mortality, than when general anaesthetics are employed.

Fall in blood pressure, formerly one of the most alarming symptoms, can be controlled by ephedrin and epinephrin, together with the Trendelenberg posture.

Postoperative nausea and headache can be easily prevented by proper attention to technique.

In our small series, too small perhaps to form definite conclusions, spinal anaesthesia by the Pitkin method, has been eminently satisfactory to the patient, relatives, nurses and doctors.

The objection which we constantly hear from patients, "I would not dread an operation if I did not have to take ether", is eliminated.

*Spinal Anesthesia Fatalities and Their Prevention. L. F. Sise. May 23, 1929.

MISCELLANY

VERMONT DEPARTMENT OF PUBLIC HEALTH
OCTOBER, 1929

Reports of communicable diseases received during the month are as follows: chickenpox 140, diphtheria 10, measles 3, mumps 5, scarlet fever 20, smallpox 43, poliomyelitis 5, whooping cough 114 and tuberculosis 20.

The Laboratory of Hygiene reports 1,482 examinations made, classified in the following manner:

Examination	
For diphtheria bacilli	200
For Widal Reaction of typhoid fever	40
For malarial parasites	0
For tubercle bacilli	251
For evidence of syphilis	299
For gonococci in pus	135
Of blood for contagious abortion in cattle	151
Of water, chemical and bacteriological	60
Of water bacteriological	114
Of milk, market	131
Of milk submitted for chemical only	6
Of foods	20
For the courts, autopsies	4
For the courts, miscellaneous	15
Autopsies to complete death returns	0
Miscellaneous examinations	56

The Division of Communicable Diseases received reports of 37 cases of gonorrhea and 26 cases of syphilis. One case of gonorrhea and two cases of syphilis reported for treatment. 117 gonorrheal and 306 outfits were sent out.

The director of the Tuberculosis Division gave illustrated talks in twenty-six towns. 9,500 copies of the Crusader were published and distributed.

The Poliomyelitis Division visited 64 patients in their homes. Sixty-six pieces of apparatus were fitted, 25 orthopedic corrections made to shoes and 15 pieces of apparatus altered or repaired. The usual fall clinics were held with an attendance of 244 patients. The vocational worker of this Division made 34 home visits. Sales have been held in several towns, the proceeds amounting to $291.01.

The nurse of the Maternity and Infancy Division visited ten towns, assisting women's clubs, meeting with Mothers' clubs and holding clinics for preschool children. In one town a three-day course in Child Care was given to a group of high school girls.

SOCIETY NOTES

The regular Fall meeting of the Windsor County Society was held at the Adnabrown Hotel, Sept. 12, 1929, where dinner was served, followed by a business meeting and several interesting papers.

Dr. Percy Bartlett, of Hanover, N. H. read a paper on Osteomyelitis, stressing particularly the importance of an early diagnosis.

C. E. Bolser, Ph.D. of Dartmouth College gave a talk on the Application of Chemistry in Diagnosis.

Dr. J. J. Boardman of Hanover, N. H. gave a talk on Obstetrics, based upon a month of observation at the Boston Lying-In Hospital.

For the year 1928-1929 one member died and for the same period three were added. Dr. Frank C. Fletcher, of Gaysville, and Dr. Stanley L. Garipay, of Springfield, were admitted to membership at this meeting.

Officers elected for ensuing year: Pres. A. L. Patch; Vice Pres. T. Gartland; Sec.-Treas. A. B. Woodman; Delegate 1 year, A. M. Cram. Delegate 2 years, Dean S. Drake. Alternate, B. A. Chapman. Censors: H. C. Jackson, A. L. Patch.

A. B. WOODMAN, *Secy.*
Windsor County Society.

The Burlington and Chittenden Clinical Society elected the following officers for the coming year on October 3:

President: Dr. W. J. Upton.
Vice-President: Dr. C. A. Ravey.
Secretary-Treasurer: Dr. R. G. Streeter.
Executive Committee: Dr. S. H. Martin, Dr. A. L. Fogg, Dr. F. K. Jackson.
Delegates for 2 years: Dr. B. D. Adams, Dr. W. G. Townsend, Dr. P. P. Lawlor, Dr. R. E. Corley.
Alternates: Dr. Sam Sparhawk, Jr., Dr. Paul K. French, Dr. N. R. Caldwell, Dr. P. E. McSweeney.

Two new members were admitted, Dr. Hiram Upton and Dr. P. D. Clarke.

The Bennington County Medical Society met Wednesday, November 20, at 8:30 P. M., at the Putnam Memorial Hospital. The annual election was held and the following officers were elected:

President: Dr. John Trotter.
Vice President: Dr. Charles S. Buchanan.
Secretary: Dr. John D. Lane.
Auditor: Dr. Frank E. Dean.
Delegate: Dr. Lucretius H. Ross.
Alternate: Dr. Edward Tobin.
Delegate: Dr. Charles S. Buchanan.
Alternate: Dr. John D. Lane.

A moving picture film demonstrating the surgical treatment of gastric ulcer was shown. The film depicted the various steps in several operations and demonstrated the subject in an excellent manner The film was loaned for the occasion by Davis and Geck, ligature manufacturers, of Brooklyn, New York.

Several fine X-ray films of the gall bladder were shown.

The Society will meet regularly once or twice a month until June next.

Bennington County Medical Society.

PERSONAL ITEMS

Drs. J. A. Wark, F. C. Angel and M. F. McGuire have returned from Detroit where they attended medical meetings.

Montpelier has two new men—both graduates of University of Vermont College of Medicine. Dr. Allan Godfrey, son of Dr. Godfrey of Morrisville, has opened an office on Main St. Dr. R. H. Bisson has practiced in Winooski for the past four years. His office is in the Perry Bldg., on State Street.

Dr. C. P. Chandler and Dr. F. W. Harriman have returned from a hunting trip to the Canadian woods.

Dr. J. P. Gifford is recuperating at his home, from a serious operation performed in Boston.

Dr. R. E. Avery has been in Boston doing special work in X-ray as has Florence Johnson, R.N., superintendent of Barre City Hospital.

Dr. and Mrs. W. E. Lazell spent two weeks in Boston and New York recently.

Mrs. E. H. Bailey wife of Dr. E. H. Bailey is a patient at Barre City Hospital.

C. H. GOYETTE, *Sec.*
Washington County Medical Society.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.

F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15481

SEVEN MONTHS' BURNING SUBSTERNAL PAIN IN A MAN OF TWENTY-NINE

MEDICAL DEPARTMENT

PRESENTATION OF CASE

BY ALLEN G. BRAILEY, M.D.*

I will read the summary prepared by the East Medical Service. A white single male American molder of twenty-nine entered September 13 complaining of burning substernal pain of several months' duration. Fourteen years before admission he contracted gonorrhea. He had acute arthritis involving the knees and ankles. His heart was supposed to be damaged at that time. He had slight dyspnea on exertion. He had no other impairment. He did hard manual work until one year before admission. Then his dyspnea and palpitation on exertion became slightly more marked. Seven months before admission he had burning pain under the sternum on exertion, with more dyspnea and palpitation. The pain went down both arms to the wrists, with a sensation of stiffness in the wrist joints. It was relieved in five or ten minutes by nitroglycerin. Recently he had had these attacks by night as well as by day. He had no edema. He had occasional rheumatic twinges in the joints.

Clinical examination showed an extremely well-developed young fellow with a slight flush on both cheeks. There was a good deal of dentistry. The heart was only moderately enlarged to the left and down, with heaving apex impulse. Thrills, both systolic and diastolic, at aortic area. Loud first sound at apex, with double murmurs. Double murmurs also at aortic area. Blood pressure 180/70. Do you want our diagnosis?

DR. CABOT: Not yet. The record says that that thrill was best felt at the apex.

DR. BRAILEY: As I remember it was felt over the aortic area.

DR. CABOT: That makes a difference. It is just the sort of correction we can get from the person who made the examination during life. There was a thrill over the aortic area. I have not heard anything yet about the second sound in the aortic area.

DR. BRAILEY: It was absent.

DR. CABOT: The absence of the aortic second sound goes along with what has been said about the thrill. There is an electrocardiogram. You have not said anything about that.

DR. BRAILEY: It showed normal rhythm, rate 100, inverted T_2 and T_3, coronary type.

DR. CABOT: The urine does not seem remarkable.

DR. BRAILEY: It was wholly normal throughout. The two Hinton and Wassermann tests were positive.

DR. CABOT: So he had two positive Hintons and Wassermanns.—I do not know whether you have the final events.

DR. BRAILEY: He was operated upon and was sent back to our ward. He was temporarily relieved of pain. On September 28 he came back and was largely free from pain, but had occasional pain similar to what he had before operation in his right arm, not in the left or very slight in the left. He had gradual onset of burning pain in the chest and along the inner surface of the right arm, only temporarily relieved by amyl nitrite. He was given pantopon and morphia with no relief whatever. He broke out into a profuse sweat. His arm grew colder. Evidently the circulation was failing. He was put on the danger list, and died the evening of September 28.

DR. CABOT: Have we any record of his leukocyte count?

DR. BRAILEY: It was 10,000, a single count taken on the 13th.

DR. CABOT: Was there a blood culture?

DR. BRAILEY: It was not done.

DR. CABOT: There was nothing at any time to suggest embolism?

DR. BRAILEY: No. He had no fever.

DIFFERENTIAL DIAGNOSIS

DR. CABOT: It seems to me that the main facts in this case fit but one interpretation, that is *coronary block*. We have the kind of pain, we have the kind of electrocardiogram, and we have a perfectly plausible idea of what might block it. In an elderly person the ordinary cause for coronary block is arteriosclerosis. Narrowing of the coronary at twenty-nine years is possible but not probable. Another cause of blocked coronary is malignant endocarditis, fragments of a vegetation on his heart valve blocking probably successive branches at different times. With the signs we have it could be endocarditis on the aortic valve. It seems to me quite plausible that there is a mass of vegetations on this valve, or an intracardiac thrombus, probably not septic in origin.

We start with the clinical picture of blocked coronary. We then ask what that might come from at his age. I can think of three plausible sources, the aortic valve, the interior of the heart and the coronary orifices; first a septic type of thrombus (endocarditis) and second, non-septic, from heart clot. I do not think temperature helps us a great deal in distinguishing between

*Senior interne on the East Medical Service.

a septic and a non-septic type of coronary thrombus. We get high temperature with bland thrombus, presumably from absorption of infarct material. The only blood count we have is 10,000, which is on the dividing line. It does not help us. We have no culture or evidence of embolism to help us in answering the question.

I think we ought to mention as less probable but not to be excluded, that the whole thing might be from coronary arteriosclerosis.

There is one further possibility; the whole thing might be syphilitic. I have left that until the last. The two positive Wassermanns, the age and sex of the patient, the valve attacked, all favor syphilis. The chief discordant note that stands out as I go over the history is that double thrill, which is not at all common in uncomplicated syphilitic aortitis. He has a high pulse pressure, 110, which of course he could have with any type of aortic lesion, but would be more likely to have with a syphilitic lesion than with a non-syphilitic. Non-syphilitic lesions cause more stenosis and throw back a smaller column of blood than syphilitic.

DR. BRAILEY: I find in my record that the thrill was at the apex and not at the aortic area.

DR. CABOT: That does not have to go at all against syphilis. It could go perfectly well with it. Syphilis of the aorta and the aortic valve, then, may perfectly well be the diagnosis. We still have to account for the thrill at the apex.

A STUDENT: He had questionable rheumatic fever.

DR. CABOT: We do not know that that was rheumatic fever. We do not know that it was not. He might perfectly well have a valvular lesion from some previous attack which does not definitely connect with the present one.

I do not see how syphilis is to be ruled out, and in many ways it seems to me to be the most probable diagnosis. I am influenced against it somewhat by the fact that in the years that I have been discussing these cases I have very seldom come out right on a diagnosis of syphilis, very often have come out wrong. I think it is a most dangerous diagnosis to make under any circumstances whatever, no matter how many things point to it. But there certainly is a good deal to point to it here.

Did he have any X-rays?

DR. BRAILEY: He had a plate of the heart on the 16th. The report is, "The transverse measurements of the heart are increased above normal limits. The curve of the left ventricle is prominent and rather blunted. The auricles do not appear enlarged. There is slight prominence of the pulmonary conus. The findings are consistent with an aortic lesion. There is no definite evidence of mitral disease." On October 1 he was sent down to see whether a rib had been cracked because of the operation. The X-ray showed a defect in the third rib.

DR. CABOT: Nothing was said about his aorta?

DR. BRAILEY: It is not mentioned in the record.

DR. CABOT: I do not doubt that he has aortic regurgitation, but I think I could have foretold all that they said about the left ventricular prominence. I do not see that they committed themselves on the most important point: does the aorta itself show disease?

We ought to say a word about that operation. What do you suppose it was?

A STUDENT: Was it cutting the spiral roots?

DR. CABOT: More probably an operation done for angina pectoris,—an operation or operations. The more I read about it the more I think that no two men do the same operation. One thing you do when a man is having pain is to make a hole in the front of his chest and cut something. Whatever you cut, it seems to help. I discussed it this September with Dr. Coffey in San Francisco, the man who has done the operation most often. I think he always cuts the same nerve. I think from what I read of it that various other nerves are cut by other operators. I can not say that is what they did here, but that is the most probable thing, I think.

PRE-OPERATIVE DIAGNOSIS

Angina pectoris.

OPERATION

Gas and oxygen. A left paramedian incision from the first to the fourth dorsal spines. The muscle was separated and one inch of the third rib was excised subperiosteally. The transverse process and the head of the rib were rongeured away. The pleura was wiped laterally off the bodies of the second and third dorsal vertebrae. This exposed the ganglionar trunk and its communicant rami from the second to the fourth dorsal. All of this portion of the trunk and ganglia were removed and all visible rami cut. The wound was then closed in three planes without drainage.

PATHOLOGIC REPORT

Stellate ganglion. The specimen consists of a sympathetic ganglion and nerve fibers. There is no evidence of disease.

FURTHER DISCUSSION

DR. CABOT: Apparently they did what you suggested and not what I thought. They did the operation to stop pain.

I do not know that I can go any further. The diagnosis with which I started is coronary block, and one can suppose the coronary is blocked at its mouth by syphilitic aortitis or in its course by a thrombus. On the whole I should think a syphilitic process would seem more probable if I had not fallen down so often on that diagnosis. I think I had better make that diagnosis even with my past experience of such diagnoses. He has also a valve lesion, of rheumatic or some other type.

A STUDENT: Would you say that to and fro murmurs are always significant of congenital patent ductus arteriosus?

DR. CABOT: No. To and fro murmurs are oftenest heard with aortic disease or mitral disease. The murmur most often recorded in patent ductus arteriosus is a murmur which does not stop at all. A to and fro murmur is like a saw. The murmur of patent ductus arteriosus is a steady "swish" going on as long as you choose to listen to it, never stopping.

A STUDENT: Is it more probable that it is rheumatic involvement of the valves rather than syphilitic?

DR. CABOT: Perhaps a little; but I should not say it makes much difference so far as I can tell. He may have both. My best guess is that he has both. He did not die of the rheumatic process, but it is the most natural thing to account for the thrill at the apex.

A STUDENT: Is not twenty-nine years early for syphilitic aortitis?

DR. CABOT: No. I have seen it a number of times earlier. There were quite a good many cases in the war in enlisted men, mostly under twenty-five.

A STUDENT: Could gonorrheal bacterial endocarditis be considered?

DR. CABOT: I cannot exclude it. But the gonorrhea was a good while ago. Most cases on record follow closely on an acute process. It is less likely than any other diagnosis we have mentioned.

A STUDENT: How often do you get a story of right sided pain with syphilitic aortitis?

DR. CABOT: It could not be called rare. I cannot give you the figures. Pain in both arms is quite common. Pain confined to the right arm occurs in less than one-tenth of the cases.

A STUDENT: After the dorsal ganglion was severed on the left side wouldn't you expect to get pain in the right?

DR. CABOT: That is a little too much for me. I can construct some hypotheses about it, but I do not know.

A STUDENT: Is this the description of the kind of pain you expect to get in angina?

DR. CABOT: It is the pain you get in coronary disease, and coronary disease can cause attacks of angina which leave easily as well as the prolonged pain of block. I do not think one can possibly call this angina. I think it is the pain of blocked coronary.

A STUDENT: Did he have water-hammer pulse?

DR. BRAILEY: There is no note about that. He had a blood pressure of 180/70.

DR. CABOT: He must have come pretty close to a Corrigan pulse.

A STUDENT: What was the clinical diagnosis?

DR. BRAILEY: The diagnosis on admission was rheumatic heart disease, mitral stenosis and regurgitation, aortic regurgitation, and angina pectoris.

DR. CABOT: Was that revised as you went on?

DR. BRAILEY: No, I think not.

INTERPRETATION OF X-RAY

The findings are consistent with an aortic lesion. There is a definite area of mitral disease.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

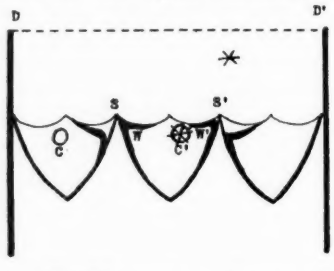
Rheumatic heart disease.
Mitral stenosis and regurgitation.
Aortic regurgitation.
Angina pectoris.

DR. RICHARD C. CABOT'S DIAGNOSES

Coronary block, due probably to syphilitic aortitis, possibly to coronary thrombosis.
Endocarditis of the aortic valve.

ANATOMIC DIAGNOSES

Luetic aortitis with aortic regurgitation and closure of the right coronary artery.



DR. TRACY B. MALLORY: I can show the lesion most easily by a diagram. The light lines represent the normal aortic valve, with the left coronary coming out at C and the right coronary about at C′. In the characteristic lesion of syphilitic aortitis the valve involvement is something like that shown by the heavy lines at S, a separation of these two valves which normally meet the aorta at a single point and a widening of the commissure here at W. The same thing occurs here at S′. There is more or less fibrosis and thickening of the cusps close to the point where they reach the aorta, but the process is primarily destructive. Approximately this lesion was found in this man, with in addition a scarring of the aorta which ran up a distance of about 3 centimeters from the valve with numerous pitted little stellate scars (as indicated by the star on the diagram) stopping quite sharply at this line, D . . . D′, more or less uniformly all the way across the aorta. One of these scars completely occluded the mouth of the right coronary artery. The entire remainder of the vessel beyond the first two millimeters was perfectly normal in appearance. In spite of the completely closed coronary there was no scarring and no infarction. There was more thickening of the valve cusps than we common-

ly see in a straight luetic aortic involvement. It made one wonder whether besides luetic there had been possibly gonorrheal or rheumatic involvement at some time in the past. His mitral valve showed perhaps a trace of fibrous thickening, but I was not convinced there was anything on it. This is distinctly against rheumatic involvement. The attack of arthritis was probably gonorrheal, and there was undoubtedly luetic infection as well at that time.

I do not know what the likelihood of recovery from gonorrheal endocarditis is. Certainly the majority of these cases behave like bacterial endocarditis and terminate fatally. We do not ordinarily prove any cases of endocarditis which recover, though septicemia is not rare in the acute stage. Dr. Joseph T. Wearn had two very interesting cases at the City Hospital in which both coronary arteries were completely occluded right at the mouth by syphilis of the aorta. In neither of those cases was there any necrosis, but an adequate blood supply was maintained presumably by the collateral circulation offered by the Thebesian veins[1]. I think something of that sort must be assumed to account for this.

DR. CABOT: Was the aorta enlarged? Do you think the X-ray man could have helped us if he had looked carefully?

DR. MALLORY: I should doubt it. There was a rather narrow zone of involvement which did not go up into the arch at all; and the arch is the point the X-ray man usually sees.

DR. CABOT: I am a little puzzled about the cause of death. I thought he was going to have an infarcted heart, but he did not.

DR. MALLORY: That seems to be characteristic of this type of coronary lesion. The cases reported by Dr. Wearn were both Medical Examiner's cases, one a man working as a seaman, the other a woman earning her living as a seamstress. Both were perfectly well until they were found dead in bed one morning for some reason. They had developed a perfectly good compensatory circulation, which allowed a good deal of activity but very suddenly failed. It seems to be the only explanation.

DR. CABOT: There is no reason to suppose the surgical operation influenced his death?

DR. MALLORY: No. If they had realized it was luetic aortitis instead of supposing it to be rheumatic, they would perhaps have hesitated to operate.

DR. CABOT: Did he have evidence of syphilis elsewhere in the body?

DR. MALLORY: Not at all.

DR. CABOT: Was there any passive congestion?

DR. MALLORY: Practically none as I remember it. The liver and spleen were both normal in size. There seemed to be just a trace of congestion in the liver, certainly no long time congestion at all, merely some acute heart failure. Six hours could account for it.

A STUDENT: Would you say that the systolic and diastolic murmurs heard at the apex were of the Austin Flint type?

DR. CABOT: No, I should say that was working Dr. Flint too hard. I should say the thrill was a profound mystery. Austin Flint did not talk about thrill, he talked only about murmurs. How the systolic and diastolic murmurs could be accounted for I have no idea. What is new to me, what I should have known from Dr. Wearn's cases, is that you can have entirely blocked coronaries with good compensatory circulation.

DR. BRAILEY: That was the thing that fooled everyone who saw the case. Dr. White heard those murmurs.

DR. CABOT: If Dr. White heard those murmurs they must have been there. It does leave it very mysterious. That is all I know how to say.

1 Joseph T. Wearn. Thebesian Vessels of the Heart and Their Relation to Angina Pectoris and Coronary Thrombosis. N. E. Jour. Med., 1928, 198:726.

CASE 15482

OCCIPITAL PAIN AND DIPLOPIA

NEUROLOGICAL DEPARTMENT

An American schoolboy thirteen years old entered February 8 complaining of pain of six weeks' duration in the back of the neck and over the occiput.

When he was five years old he had similar pain over the neck and occiput accompanied by visual disturbances lasting two or three days at a time. He was given glasses. The condition gradually improved, although he had had the pain at intervals ever since that time. The autumn before admission he was apparently perfectly well and played football. After this he began to be less well and had no desire to go out. For the past six weeks he had had dull ache over the neck and occiput accompanied sometimes by stiffness of the neck, practically constant throughout the day and making it difficult for him to go to sleep. For six weeks he had had diplopia, dimming of vision, ptosis of the eyelids and increasing fatigability of the eyes. Six weeks ago he had obstinate constipation for a short time. For the past two weeks it had been very troublesome, his bowels moving only once in three or four days with cathartics. He found that on walking he tended to turn to the left, especially during the attacks of pain and more when his eyes were closed. He had had some ataxia; no vertigo. Often on rising after sitting he fell. Lately his hearing had been poor, especially in the left ear.

His family history is not significant.

He had pneumonia three and again four years before admission. He was a mouth breather—probable nasal obstruction. He had occasional sore throats, and had been advised to have tonsillectomy. When constipated he had abdominal pains. He was occasionally nauseated and had

rarely vomited. During the past two years he had grown tremendously. Two months before admission he weighed 165 pounds, his best weight. He had reached the seventh grade in school.

Clinical examination showed a robust boy with flushed cheeks lying inert. He was conscious and oriented but obviously his mental processes were retarded and interfered with. Temperature normal. (It had been subnormal, according to reports.) His speech was slow and a little thick, with marked slurring of test words. *Tâche cérébrale* appeared in a minute and lasted twenty minutes or longer.

Neurological examination. He said he could smell and taste normally. Vision of both eyes poor, right eye 20/40, left eye 20/200. The fields appeared normal to gross tests. Marked choking of the dises, right eye 2.5 diopters, left eye 3. Limitation of conjugate movements in all directions except downward. The eyes moved about 30° from the center. There was no diplopia at present and no lid lag, ptosis, strabismus, nystagmus or exophthalmos. Both eyes were slightly prominent. The winking reflex was reduced. The pupils were equal, a little dilated, reacted to light and distance. The right pupil was slightly oval and not absolutely regular and its reaction was not quite so quick to light as that of the left. Corneal reflexes prompt and equal. Motor and sensory branches normal. 7th nerve. Suggestion of weakness in lower right side when patient showed his teeth. When he wrinkled his face the brow was slightly flatter on the right. It could not be said definitely however that there was any central paralysis. He heard watch tick at two and a half feet on the right side, at six inches on the left side. He had himself noticed that in the last six weeks he heard better on the left (sic) side. The raphe did not deviate, but the uvula was pulled over to the right. Bilateral reflex was absent, gag reflex absent or much diminished. Right arm definitely weaker than left. Right grip weaker. He said it had always been so. Finger-to-finger, finger-to-nose, and heel-to-knee normal. No past pointing or adiodokokinesia. No Romberg. He walked unsteadily but with no definite deviation. He thought he had a tendency to list toward the right, but it was not noticeable. Sense of direction good with eyes shut. Stiff neck. Not much pain. Arm-jerks, knee-jerks and ankle-jerks normal and equal. Abdominals absent. Cremasterics normal. No Oppenheim, Gordon or Babinski. Definite plantar flexion of both feet.

Urine and blood not recorded.

Before operation temperature 97.3° to 99.4°; pulse and respirations normal.

X-ray examination of the sella turcica and calvarium showed no definite variation from the normal.

On February 9 examination showed pronounced vertical nystagmus upon looking up. Motion of the eye was limited in all directions. Right facial weakness—central (?) weakness of the right sixth nerve. The right knee-jerk was greater than the left and the right leg showed greater tonus than the left. The patient was slightly more sluggish.

February 10 a combined bilateral ventricular and lumbar puncture was done. A punch specimen of brain tissue was taken and air was injected.

February 12 the only reflex which could be elicited upon stroking the abdomen was a small one in the left upper quadrant. To the left of the raphe the palate appeared to rise slightly higher than to the right.

February 13 operation was done. The patient made a fairly good postoperative recovery. He was rather drowsy but was easily roused, answering questions intelligently. His speech was rather thick but not markedly different from what it was before operation. February 16 cerebrospinal fluid was leaking. A lumbar puncture was done, with withdrawal of about 30 cubic centimeters of blood stained fluid under increased pressure. Reënforcing skin sutures were placed at the site of the leak. The leakage continued, however. February 19 40 cubic centimeters more of blood stained fluid was withdrawn under increased tension. On the 22nd he seemed brighter than at any time since the operation. Two days later he began to show some aphasia. Repeated restrappings of the wound and reënforcing sutures failed to stop the leak. March 1 there was an indurated reddened area on the right shoulder and right side at about the ninth rib. The temperature began to rise. The patient was very stuporous. The following morning he choked on food and did not swallow. He did not respond to questions. The sclerae and conjunctivae were injected. The pulse and respirations were rapid. There was slight cyanosis of the hands. The upper lobe of the right lung showed beginning consolidation. That afternoon he died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

The presenting symptoms in this case make us think at once of cerebral or cerebellar tumor. The fact that he has so much constipation, that in walking he tends to turn to the left, that he evidently has difficulty in standing when he first rises from a chair, and the difficulty with his hearing, all tend to support this suggestion. His family history and past history do not help us.

NOTES ON THE PHYSICAL EXAMINATION

In the clinical examination the presence of some cerebral lesion is further evidenced by the

mental condition, the speech, and the *tache cérébrale.* Choking of the discs, limitation of eye movements, abnormal pupillary reactions, deafness in the left ear, weakness of the right arm, stiff neck, plantar flexions in both feet, with the later occurrence of nystagmus, weakness of the right sixth nerve, all go to confirm the diagnosis suggested by the symptoms mentioned at the beginning of the record.

DIFFERENTIAL DIAGNOSIS

I see no reason to doubt that he has cerebral tumor. Though we have no report of the lumbar fluid obtained by puncture, I have no doubt that it was normal so far as any evidence of *meningitis* was concerned. There is no evidence of a *cerebral abscess* either in the history, the blood, or the temperature. He is too young for a *vascular brain lesion.* If the diagnosis then is cerebral tumor, what can we say as to its localization? Presumably it is on the left side, as the eye and ear symptoms seem to be on that side, and the weakness of the right arm together with the increased right knee-jerk and greater tonus on that side also point to a left-sided lesion. The terminal event was apparently a right-sided pneumonia.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Glioma, left parietal region.

DR. RICHARD C. CABOT'S DIAGNOSIS

Cerebral tumor, left side.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Spongioblastoma of the left parietal region.
Acute purulent meningitis.

2. *Secondary or terminal lesions.*

Operation wound—left parietal decompression—hernia of the brain.
Bronchopneumonia.

3. *Historical landmarks.*

Precocious sexual development.
Metaplastic bone formation in adrenals (bilateral).

DR. TRACY B. MALLORY: Post-mortem examination confirmed the chief clinical diagnosis of a tumor in the left parietal region. It was about 4 centimeters in diameter, lay anterior and external to the posterior horn of the lateral ventricle and involved the posterior part of the lenticular nucleus, the posterior half of the posterior limb of the internal capsule and the outer, posterior portion of the optic thalamus.

Microscopic examination showed it to consist of relatively undifferentiated glial cells which with gold sublimate stains could be identified as unipolar and bipolar spongioblasts. This identifies it as a relatively rapidly growing type of glioma which offers an unfavorable prognosis for any type of palliative operation. As so often happens in these cases, herniation of the brain had occurred. The wound failed to stand the strain and broke open, with a terminal meningitis. A very slight bronchopneumonia was also present.

Another very interesting finding, unfortunately not brought out in the clinical record, was an unusually precocious sexual development. Although he was only thirteen years of age the genitals were of adult proportions, the testes showed active spermatogenesis, and the secondary sexual characteristics such as pubic hair were of the adult type. A most unusual finding was symmetrical calcification of part of the medulla of each adrenal gland. A portion of this calcified material had been organized into bone between the trabeculae of which was found fatty marrow. Precocious sexual development is of course common in adrenal tumors in childhood. Whether the unusual lesions present in this boy's adrenals can be connected with the degree of sexual development is an interesting field for speculation.

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NOTES ON THE PHYSICAL EXAMINATION

In the clinical examination the presence of some cerebral lesion is further evidenced by the

mental condition, the speech, and the *tache cérébrale*. Choking of the discs, limitation of eye movements, abnormal pupillary reactions, deafness in the left ear, weakness of the right arm, stiff neck, plantar flexions in both feet, with the later occurrence of nystagmus, weakness of the right sixth nerve, all go to confirm the diagnosis suggested by the symptoms mentioned at the beginning of the record.

DIFFERENTIAL DIAGNOSIS

I see no reason to doubt that he has cerebral tumor. Though we have no report of the lumbar fluid obtained by puncture, I have no doubt that it was normal so far as any evidence of *meningitis* was concerned. There is no evidence of a *cerebral abscess* either in the history, the blood, or the temperature. He is too young for a *vascular brain lesion*. If the diagnosis then is cerebral tumor, what can we say as to its localization? Presumably it is on the left side, as the eye and ear symptoms seem to be on that side, and the weakness of the right arm together with the increased right knee-jerk and greater tonus on that side also point to a left-sided lesion. The terminal event was apparently a right-sided pneumonia.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Glioma, left parietal region.

DR. RICHARD C. CABOT'S DIAGNOSIS

Cerebral tumor, left side.

ANATOMIC DIAGNOSES

1. *Primary disease.*

Spongioblastoma of the left parietal region.
Acute purulent meningitis.

2. *Secondary or terminal lesions.*

Operation wound—left parietal decompression—hernia of the brain.
Bronchopneumonia.

3. *Historical landmarks.*

Precocious sexual development.
Metaplastic bone formation in adrenals (bilateral).

DR. TRACY B. MALLORY: Post-mortem examination confirmed the chief clinical diagnosis of a tumor in the left parietal region. It was about 4 centimeters in diameter, lay anterior and external to the posterior horn of the lateral ventricle and involved the posterior part of the lenticular nucleus, the posterior half of the posterior limb of the internal capsule and the outer, posterior portion of the optic thalamus.

Microscopic examination showed it to consist of relatively undifferentiated glial cells which with gold sublimate stains could be identified as unipolar and bipolar spongioblasts. This identifies it as a relatively rapidly growing type of glioma which offers an unfavorable prognosis for any type of palliative operation. As so often happens in these cases, herniation of the brain had occurred. The wound failed to stand the strain and broke open, with a terminal meningitis. A very slight bronchopneumonia was also present.

Another very interesting finding, unfortunately not brought out in the clinical record, was an unusually precocious sexual development. Although he was only thirteen years of age the genitals were of adult proportions, the testes showed active spermatogenesis, and the secondary sexual characteristics such as pubic hair were of the adult type. A most unusual finding was symmetrical calcification of part of the medulla of each adrenal gland. A portion of this calcified material had been organized into bone between the trabeculae of which was found fatty marrow. Precocious sexual development is of course common in adrenal tumors in childhood. Whether the unusual lesions present in this boy's adrenals can be connected with the degree of sexual development is an interesting field for speculation.

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POST-TRAUMATIC NEUROSES

NEW light was thrown on the post-traumatic neuroses by our vast experience of this type of disease during the World War. The so-called "shell shock" patients, of which there were a large number, gave us material for study such as we never had had previously and the conception of what a post-traumatic neurosis could be became really defined. The importance of these conclusions is especially brought out in dealing with patients seeking compensation for occupational injuries or those who seek compensation through litigation for injuries through accidents, not coming under our Industrial Accident Board. The Accident Board group is, perhaps, the most troublesome of all to the physicians and others who are attempting to carry out the provisions of the Industrial Accident Law and any new ideas which will give an even better understanding of these patients are of great value. Recently there has appeared an article by Drs. Schaller and Somers of San Francisco* in which they point out, after a careful study of a large number of cases, that there is always a "precipitation point" for the neurosis, which may occur early or late after the physical trauma has taken place. For instance, they cite a case of a man who had a laceration of his arm, hospitalized for two months. The patient's efficiency, from the working man's point of view, during this period, of course was nil. At the end of this time the employee returned to light work and for a number of months increased his efficiency to about 70 per cent. In the meantime there developed ideas of improper treatment and an idea that the severity of the injury was not, in the opinion of the patient, completely recognized by those who had treated him. There was also a visible deformity. Eleven months after the injury he was not able to carry on his work and gradually fell off so that at the end of two years he was again unable to do any work and had a completely developed post-traumatic neurosis. The "precipitation point", which led to his loss of efficiency, occurred nearly a year after his injury and six months later he could do no work.

This single case, as well as the many others in the article referred to, clearly illustrated by charts, points to the extreme importance of the period of some months *after* the injury, in which patients of this type are highly suggestible. There is no question, in most of these cases, of malingering, but the fact must be faced that these patients developed hysterical symptoms because of their increased suggestibility. Once the patient has the idea that treatment has not been adequate, or that the injury was more serious than anticipated at the start, or his friends call attention to the deformity and insist that he will never be as good as he was before, there develops in his mind the condition which we call a neurosis.

The "precipitation point" may be even longer after the initial injury and, moreover, it may be a factor which ultimately causes entire relief so that the patient returns to a normal efficiency, the reverse of the situation already cited. A good illustration of this is the second case given by Drs. Schaller and Somers. Briefly, in 1918 a man had a fracture of the arm; he was in the hospital twelve days and the fracture was set twice and finally plated; there was a traumatic radial palsy, which responded slowly to treatment, so that at the end of a year he was able to go back to part-time work. He kept at this for a number of months, but he was worried and rather introspective about his condition and later got the idea from somewhere that one of the screws in the bone plate was loose, the "precipitation point" of the neurosis. His work fell off and he was unable to do anything. A few weeks later the arm plate was removed; the patient almost immediately returned to work at a full-time level and has continued to do so ever since. Here, the post-traumatic neurosis had its

*Schaller, W. F. and Somers, M. R. Psychogenic Factors and Precipitation Point in the Post-Traumatic Neuroses. J. A. M. A., 93:967, 1929.

"precipitation point" at the time when the idea of the loose screw in the plate developed. The removal of the plate was sufficient to break up the mental state associated with this condition of hyper-suggestibility and the man was completely cured.

The same type of reaction, of course, occurs occasionally when compensation is effected or the patient is no longer receiving compensation for his injury. There are many examples of this type of reaction, which cannot be put upon a basis of malingering but are really part of the neurosis and a normal reaction to the situation when the suggestion of compensation is stopped. One frequently sees this type of reaction without special litigation. Schaller and Somers give a case in which a man inhaled some gas fumes, was out of work for nineteen days, returned to light work, which he could not do efficiently, and was discharged. His discharge precipitated a post-traumatic neurosis and he went downhill within the course of a few months so that he could not do anything. Compensation, in the meantime, had been carried on. It was decided, after more than a year, to stop the compensation. The man rapidly recovered and later entered the U. S. Army. This type of reaction was so common during the War that certain Army officers among the allied forces would not pay any compensation for post-traumatic neuroses, a preventive measure which probably saved many soldiers from this disease.

Most writers on this subject agree that the so-called "split compensation", as compared with "lump compensation", is a serious factor in prolonging the post-traumatic neurosis. Even a small amount of money which is sufficient for a person to live on, paid week after week, is enough to keep the neurosis going. Many insurance companies would be saved large amounts of money could they visualize the "precipitation point" and stop "split compensation" at this time.

One must recognize, also, the period of suggestibility *before* the "precipitation point". Care should be taken not to talk too much to the patient about his injury, remembering that he probably has a very false idea in regard to the anatomy and physiology of his own body and more important still, he is almost sure to be more suggestible than in his normal condition of health. We are reminded of the old story of the medical student who asked his senior what was the secret of the great man's success. The answer came back quickly, "Don't talk too much." It is said, also, that there was a very successful practitioner who saw large numbers of patients, through his many years of practice, and whose main therapy consisted of silence plus a nod of the head. Once the neurosis has developed, it is likely to be a long illness unless strenuous efforts are made to cut it short. It seems obvious that the quickest method of cutting the neurosis short is to be watchful during the period of suggestibility, visualize the "precipitation point" and pay a lump compensation for the injury involved, even if this amount appears to be relatively large at the time.

THE NEW SHRINE

THE hope for a miracle—a doubting faith in miracles—is eternal in the human mind. Born into suffering as the sparks fly upward, man is ever in search of the short cut that will relieve him of his pain and release him forever of the necessity of patience in working out his destiny. It is inborn in us, this desire to cut at one stroke the Gordian knot, to throw off our mortal burdens and to stand forth free.

Miracles furnish the glamour of the Testaments, and the lapse of nineteen hundred years has rid us of the necessity of trying to interpret them on a rational basis. So many centuries are piled up upon them that it is futile for us to try and apply to them the measure of scientific accuracy. Man's memory is too short; his observation is too obtuse in periods of emotional stress to be entirely reliable.

New miracles have been quoted and new shrines have been established, however, through all the ages. We now, in this mechanical age, have before us the spectacle of hundreds of thousands of afflicted believers—and of idle curiosity seekers—flocking to the nearby grave of a youthful priest who died almost exactly sixty years ago. The rumor was set on foot that at this grave miracles of healing had taken place, and the stampede began.

Modern miracles of healing generally resolve themselves into two classes—the healing of those who had only an imaginary disease to begin with, and the imaginary cure of those afflicted with organic disease. The permanency of either of these cures may be speculated on; presumably the first may occasionally be of lasting value, although one is reminded of Billy Sunday's reference to the bath—its value is not wholly destroyed by the fact that it needs an occasional repetition.

Will the grave of this holy youth become a shrine which will permanently grip the imagination of the emotional masses and stimulate their belief for years to come? Will he become another St. Ann de Beaupré? This we cannot answer except to say that the emotional masses are fickle, and that today, as little as at any time, are we building on permanent foundations.

Sometime a cold analysis of these modern miracles will occur to the miracle seekers, and their faith may be turned into doubt. There is the supposed instance of a child who had been blind since birth and who, when sight was given to him, read easily the legends on the tombstones. How had he learned to read? Perhaps this was the crowning achievement of his miracle. There is the instance of the deaf mute

child who talked intelligibly when speech was first granted him. How had he learned to talk? Speech was a new experience with him at that instant.

The amazing thing is to realize what a little distance the human mind has travelled since the mysticism of the middle ages; what a short step it is back to the days of Salem witchcraft. One can almost believe that the terror of that period might be repeated today.

HOW FAR SHALL THE PRESENT CODE OF ETHICS GOVERN PHYSICIANS?

Medical ethics as adopted by the medical profession for centuries has placed that profession in an enviable position. We often hear among laymen expressions of appreciation of our standards which seem to place medicine on a high plane, and also that in service to mankind physicians are not excelled by members of other professions. No one of us who is vitally concerned will contradict such statements, nor will we refuse to accept the encomium. There is no question of the disinterestedness and integrity of purpose among the vast majority of medical men. "The patient must be served."

May not the code of ethics be made more elastic in meeting the wishes of the patient under some circumstances without degrading the doctor? Fair-minded physicians and many laymen often have encountered situations in which a so-called spirit of professional ethics has been distinctly distasteful and even detrimental to the welfare of the patient. For example, when the code makes it practically impossible under some circumstances for the layman to employ the doctor of his choice, because of the disinclination of the attending physician to make the way clear for another practitioner to take over the case or to provide the special consultant suggested by the patient, what is the remedy? Under such circumstances may it be that the interest of the doctor and not that of the patient is the obvious explanation in the mind of the patient. It is certainly true that the more independent laymen are gradually taking things into their own hands and hiring whom they please at any time. And why should they not? If a physician cannot retain the confidence of his patient and finds that the patient wishes to consult another doctor, is it obligatory on other practitioners to refuse the services and force the patient to remain under the care of one who for various reasons has failed to inspire confidence?

A physician should be actuated by a desire to serve that patient rather than himself and take the first step in freeing the patient from constraint. Much would be gained by our taking such initiative not only directly by removing the cause of friction among colleagues, but indirectly by restoring confidence in the attitude of the individual physician.

It should be recognized that no physician has a mortgage on the ill health of any person and that the public should be allowed to select medical advisers without fear of censure or criticism by members of the profession. Furthermore, let it be understood that physicians may treat patients who come voluntarily to them without the expectation of being made aware of jealousy or envy on the part of other doctors.

It would seem unnecessary even to refer to such situations as suggested above if illustrations were uncommon. Unfortunately the qualities of candor, fairness and the spirit of the golden rule do not always prevail in the human heart.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

White, Franklin W. S.B., M.D. Harvard 1895. Instructor in Medicine, Harvard. Visiting Physician, Boston City Hospital. His subject is: "Observations on the Healing of Gastric Ulcer." Page 1075. Address: 322 Marlborough Street, Boston.

Cole, Lewis Gregory. M.D. Columbia University College of Physicians and Surgeons, 1898. Roentgenologist, French Hospital; Fifth Avenue Hospital; Harbor Hospital, New York City; Bronxville Hospital, Bronxville, N. Y.; and United Hospital, Portchester, New York. His subject is: "Anatomical Factors Predisposing to Corporic Ulcer." Page 1081. Address: 36 East 61st Street, New York City.

Lund, Fred B. A.B., A.M., M.D. Harvard 1892. F.A.C.S. Surgeon-in-Chief, Carney Hospital. His subject is: "The Importance of Medical Care and Consultation in Cases of Gall Bladder Disease." Page 1089. Address: 319 Longwood Avenue, Boston.

Rehfuss, Martin E. M.D. University of Pennsylvania 1909. Assistant Professor of Medicine, Jefferson Medical College. His subject is: "The Motor Mechanism Involving the Antrum, Pylorus, and Duodenum." Page 1095. Address: 16th and Spruce Streets, Philadelphia, Pennsylvania.

Anderson, George R. M.S., M.D. University of Vermont College of Medicine 1898. F.A.C.S. Surgeon-in-Chief, Brattleboro Memorial Hospital. Surgeon, Boston and Maine Railroad, Austine and Brattleboro Retreat Hospitals. President, Vermont State Medical Society. Vice President at the time of delivery of this address. His subject is: "Some Observations on Spinal Anesthesia." Page 1102. Address: Brattleboro, Vermont.

MISCELLANY

THE ANNUAL AWARD BY THE *PICTORIAL REVIEW*

The custom of this organization is to make an annual award of $5,000 to some American woman who has performed distinguished service in the field of American art, science or letters.

Dr. Florence R. Sabin was the recipient of this award for the year 1929 because of her contributions to medical science which include a complete study of the nerve-centers; the discovery of the origin and processes of the lymphatic system; the discovery of the development and processes of the blood-cell; the discovery of the functions of the monocyte, that white blood-cell which bears so directly upon the study of the tubercular germ and its effect on the human system. These discoveries were made by Dr. Sabin in her own original research work.

With her associates she has had much to do with the analysis of the tuberculosis germ and has engaged in studying the possibilities of a cure for this disease.

Dr. Sabin was the first woman to be admitted to and graduate from Johns Hopkins Medical University and to be accepted as an intern, as well as the first woman to be a member of its Staff where she was Professor of Histology. She has been recognized in European research laboratories, associated with men in Italy, Leipzig and Heidelberg. She was the first and is today the only woman member of the National Academy of Science.

Dr. Simon Flexner has paid her the tribute of declaring that she is the greatest woman scientist of all times.

The members of the award committee are: Senator Arthur Capper of Kansas, Mr. Otto H. Kahn of New York, Mrs. Mary Roberts Rinehart, Mr. Clark Howell of the Atlanta Constitution, Miss Ida Tarbell, Miss Mary Woolley, president of Mt. Holyoke University; ex-Senator James D. Phelan of San Francisco, Mr. Augustus O. Thomas, president of the World Conference on Education; Mr. Carl Sandbury, Mr. John Farrar, Literary Editor; Mrs. Charlotte Perkins Gilman, Madam Louise Homer, Dr. Henry Goddard Leach, editor The Forum; Mr. Edwin Markham, Mrs. Henry W. Peabody, Mrs. Edith Nourse Rogers, member of the House of Representatives; Mrs. John F. Sippel, president of the General Federation of Women's Clubs, and Mr. Walter L. Clark.

NEW CENTER FOR INDUSTRIAL SURGERY AND TEACHING IN NEW YORK

Announcement was made November 19 by the Boards of Directors of the New York Post-Graduate Medical School and Hospital at 20th Street and Second Avenue and the Reconstruction Hospital at 395 Central Park West, that the two institutions had been consolidated for the continuance of the purposes of each and more especially, for the advancement of traumatic and reconstructive surgery, with teaching the outstanding function of the merged institutions.

In statements issued by Mr. Allen Wardwell, President of the Reconstruction Hospital; Dr. James F. McKernon, Honorary President and Dr. Edward H. Hume, Executive Vice-President, of the Post-Graduate, the consolidation was hailed as opening up a new era for both institutions in treatment and teaching in the special field of traumatic surgery, especially because of the increasing number of serious accidents incident to modern industrial life.

PIONEER WORK OF RECONSTRUCTION HOSPITAL

The Reconstruction Hospital, according to the announcement, was a pioneer in traumatic surgery. The institution was originally established as the New York Red Cross Hospital founded during the Spanish-American war, when the late Clara Barton was head of the American Red Cross. This was the only hospital established in this country by the Red Cross which later discontinued the policy of erecting hospitals of its own, in this country. The New York institution subsequently became the Park Hospital in 1902. During the Spanish American War, the hospital sent a unit of doctors and nurses to Siboney in Cuba to care for casualties among the American forces. During the World War, the hospital cared for large numbers of wounded men in the Naval forces. In 1918, Dr. W. Gilman Thompson, since deceased, established the Clinic for Functional Re-Education. Its chief purpose was to carry into civil life the technique and knowledge which had been developed during the war for restoring wounded and injured men. The growing number of industrial accidents and diseases was found to offer an important field for such service in civil life. The Park Hospital and the Clinic for Functional Re-Education, were combined in 1922 with the Demilt Dispensary, dating from 1851, the consolidated Reconstruction Hospital thus being a consolidation of three important institutions. The land on which the present Reconstruction Hospital stands was donated by the late William T. Wardwell, the father of the present President of the Reconstruction Hospital.

PLANS FOR THE FUTURE

It is now proposed by means of the merger between the Reconstruction and the Post-Graduate institutions, to develop such further opportunities for research and teaching in traumatic surgery and industrial diseases, as to provide a center in which instruction may be given to surgeons in the most modern methods of caring for injured persons and restoring them to the working community. The laboratory facilities provided by the combined equipment, it was said, will make it possible to investigate with precision the nature and treatment of the increasing number of perplexing conditions growing out of modern industrial life.

JOINT BOARD OF DIRECTORS

The consolidated institution will be operated by a Board of Directors which will include the present

Directors of the Post-Graduate and nine members of the present Board of Reconstruction. The Post-Graduate Directors are Robert E. Allen, Vincent Astor, Edgar H. Boles, Henry D. Chapin, M.D., Clarence Dillon, William Fahnestock, William V. Griffin, Edward H. Hume, M.D., Ludwig Kast, M.D., James F. McKernon, M.D., Dave Hennen Morris, Rev. Karl Reiland, D.D., George T. Slade, Martin Taylor, and James P. Warburg.

The Reconstruction representation will include: Thomas Crimmins, Robert C. Hill, Ralph S. Stubbs, George A. Vondermuhll, and Allen Wardwell.

WEEKLY HEALTH INDEX

Telegraphic returns from 65 cities with a total population of thirty million for the week ending November 16 indicate a mortality rate of 11.5 as against a rate of 12.4 for the corresponding week of last year. The highest rate (17.8) appears for Richmond, Va., and the lowest (6.1) for Paterson, N. J. The highest infant mortality rate (131) appears for Knoxville, Tenn., and the lowest for Cambridge, Mass., and Lynn, Mass., which reported no infant mortality.

The annual rate for 65 cities is 13.0 for the forty-six weeks of 1929, as against a rate of 12.8 for the corresponding weeks of 1928.

THE STATUS OF A SUIT FOR ALLEGED MALPRACTICE

The case of Elizabeth Malony vs. Dr. E. G. Brackett and Dr. M. N. Smith-Petersen entered in November 1926 after several delays was brought to trial on October 7, 1929 before his honor Judge Sisk. After trial of six days before a jury the judge gave the defendants a directed verdict.

The counsel for the plaintiff has appealed to the Supreme Court so that considerable time will elapse before final disposition.

NEW YORK'S HEALTH CAMPAIGN

Over a million people will have health examinations, hear lectures on preventing disease, and be reminded through booklets and posters of the Health Campaign of the Five County Medical Societies of New York if the efforts of 750 groups of social and welfare organizations which are backing the Campaign are successful. Y. M. C. A., settlement-houses, baby health stations, missions, churches, clinics, health centres, and the Salvation Army are the agencies involved.

Community Councils of Social Agencies, chief among which are the Clinton Neighborhood Conference, the Kips Bay Neighborhood Conference, the Chelsea Council Social Agency, the North and the East Harlem Social Agencies, the Lower West Side, and the Lower East Side Community Councils, are the leaders in the movement to reach the public by the direct contact of social and welfare workers with the community members.

Visiting nurses will urge the examination, speakers will address meetings of Mothers' Clubs, and motion pictures showing the making of a health examination will be exhibited, in addition to the distributing of printed matter.

The Parent-Teachers Associations, the public libraries and the New York League of Mothers' Clubs are also co-öperating in the dissemination of health examination literature and propaganda.

PSYCHOLOGICAL ASPECTS OF MEDICINE STRESSED

Emotional Attitude Patient May be Cue to Nervous Diseases, Says Lecturer at Academy Medicine Meeting

Are you indifferent or coöperative during a health examination? Is your gait regular or jerky? Is your speech controlled or breathless? Your pronunciation clear or jumbled?

Though the answer to these questions may not be significant of anything important to the layman, they reveal a whole mine of information to the doctor. These psychological indicators as cues to mental and nervous diseases were stressed by Dr. Louis Casamajor, Professor of Neurology at Columbia University, in a lecture on "Neurology and Psycho-Neurology in Relation to Period Health Examination," delivered to the doctors of the New York County Medical Society, at a meeting held November 18 at 8:30 P.M. at the Academy of Medicine, 2 East 103rd Street. It is not necessarily the job of the doctor to be a psychiatrist, said Dr. Casamajor, but the observations he can make as to the mental and nervous condition of his patients in the course of a health examination will assist him in the better interpretation and handling of that patient's physical condition. Such a matter as complete indifference to the examination may be the first cue to an unsuspected case of developing dementia praecox, said Dr. Casamajor. On the other hand, people who come to a doctor, believing themselves to be suffering some mental aberration, may be merely misled by their own analysis. "People may come to you," said Dr. Casamajor, "claiming to have lost their memory. Ask them what sort of things they forget, and if they are able to answer that question, it is not a matter of memory, but merely of attention."

Other speakers were Dr. John M. Wheeler, Professor of Opthalmology at Presbyterian Hospital Medical Centre, who spoke on "The Eye in Relation to Periodic Health Examination"; Dr. Westley M. Hunt, Clinical Professor of Otology at University and Bellevue Medical College, on "Otolaryngology in Relation to Periodic Health Examination"; and Dr. Henry S. Dunning, Professor of Oral Surgery at Columbia Dental College, on "Stomatology in Relation to Periodic Health Examination."

The program was introduced by Dr. George W. Kosmak, First Vice-President of the Medical Society of the County of New York. A specially prepared motion picture showing graphically the details of

conducting the health examination, made under the auspices of the Committee on Health Examination of the Medical Society of the County of New York, preceded the lectures.

Dr. Frederick W. Bancroft presided over the meeting.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS, OCTOBER, 1929

GENERAL PREVALENCE

During the month of October the usual number of communicable diseases was reported.

Diphtheria continues to run considerably above the expectancy. The rates for lobar pneumonia and chickenpox are likewise slightly high.

The reported incidence of pulmonary tuberculosis after several low months almost exactly equals the prosodemic index. Whooping cough and scarlet fever were likewise within endemic limits. Similarly, the reported incidence of german measles, mumps, gonorrhea and syphilis was within the endemic limits.

Typhoid fever set a new low record for October, this being the fifth time this year that a new low monthly record has been reached by this disease.

The reported incidence of poliomyelitis, measles, influenza, and non-pulmonary tuberculosis was likewise low.

RARE DISEASES

Anterior poliomyelitis was reported from Boston, 8; Cambridge, 5; Everett, 1; Fall River, 1; Georgetown, 1; Hingham, 1; Lowell, 2; Lynnfield, 1; Malden, 1; Newton, 1; Salem, 2; Southbridge, 1; Springfield, 1; Wakefield, 4; Waltham, 1; Winthrop, 1; total, 32.

Anthrax was reported from Salem, 1; total, 1.

Encephalitis lethargica was reported from Boston, 1; Framingham, 1; Marlboro, 1; Warren, 1; Worcester, 1; total, 5.

Epidemic cerebrospinal meningitis was reported from Chicopee, 1; Holyoke, 2; Leominster, 1; Medford, 1; Newton, 1; Worcester, 3; total, 9.

Septic sore throat was reported from Arlington, 1; Boston, 6; Cambridge, 1; Easton, 1; Fall River, 1; Haverhill, 1; Pittsfield, 1; Worcester, 1; total, 13.

Tetanus was reported from Fitchburg, 1; Gloucester, 1; Worcester, 1; total, 3.

Trachoma was reported from Boston, 1; Cambridge, 1; Gardner, 1; Hanson, 1; Lynn, 1; New Bedford, 1; total, 6.

MONTHLY REPORT OF CERTAIN COMMUNICABLE DISEASES IN MASSACHUSETTS

	Cases in Entire Population				Case Rates per 100,000 Pop.		
	Oct. 1929	Oct. 1928	Prosodemic Index	Epidemic Index	Oct. 1929	Oct. 1928	Expected Rate***
ALL CAUSES	4544	4597	-	-	103.7	106.3	-
Ant. Poliomyelitis	32	50	66*	.5**	.7	1.2	1.5
Diphtheria	436	413	358*	1.2**	10.0	9.5	8.2
Measles	294	796	633*	.5**	6.7	18.4	14.5
Pneumonia, Lobar	281	268	200*	1.4**	6.4	6.2	4.6
Scarlet Fever	632	532	758*	.8**	14.4	12.3	17.3
Tuberculosis, Pul.	414	419	415*	1.0**	9.5	9.7	9.5
Typhoid Fever	37	39	51*	.7**	.8	.9	1.2
Whooping Cough	462	336	570*	.8**	10.5	7.8	13.0
Chicken Pox	501	421	-	-	11.4	9.7	-
German Measles	22	21	-	-	.5	.5	-
Gonorrhea	436	439	-	-	10.0	10.2	-
Influenza	16	32	-	-	.4	.7	-
Mumps	222	149	-	-	5.1	3.4	-
Syphilis	153	128	-	-	3.5	2.9	-
Tuberculosis, O.F.	53	48	-	-	1.2	1.1	-

* This Index is an attempt to estimate the number of cases based on the trend during the past years which can be expected to occur, and is for the purpose of comparison with the number of cases which actually did occur.

** This ratio expresses how prevalent the disease is compared with the index mentioned above; 1.0 indicates that the actual number of cases equals the expected number. A larger number means a greater prevalence, and a smaller number a lesser prevalence than expected. Thus, 2.0 would indicate twice the expected number of cases, and .5 half the expected number of cases. The method used to determine the indices is described in the August 18, 1927 issue of the Boston Medical and Surgical Journal.

*** Calculated from the Prosodemic Index.

CORRESPONDENCE

FRIEDREICH'S ATAXIA—MISTAKEN DIAGNOSIS

To the Editor of the NEW ENGLAND JOURNAL OF MEDICINE:

A mistake in diagnosis, if corrected many years later, is often a subject of profitable brief review, as illustrated by the following incident:

On November 6, 1915, G. E. G., a boy 8 years of age, from Fall River, Mass., was seen in the Nerve Room, Out-Patient Department, Massachusetts General Hospital (O. P. D. No. 283969 and House No. 205179), whose chief complaint was weakness of the legs and headache. His illness had begun four or five weeks before, first with headache, followed by uncertainty of gait; he stumbled about the house and frequently fell. There was no pain or paresthesia. A week before entrance to the Hospital he had been a patient in the Fall River Hospital for two days, where a diagnosis of Friedreich's ataxia was made. The patient came from a healthy family and his past history indicated that he had had no serious illness or accident up to the time of being seen.

The most striking features brought out by the examination were marked swaying and falling in the Romberg position, even with his eyes open, and an extremely ataxic gait. He walked with a reeling motion; the deep reflexes of the legs were absent; there was no ankle clonus and the plantar response was normal. The arm reflexes were barely obtained and there was a moderate amount of ataxia of the arms. His pupils reacted normally to light and accommodation. The ataxic gait plus the loss of reflexes suggested to the minds of those who saw him first that they were dealing with a case of Friedreich's ataxia. The headaches, however, in spite of the absence of changes in the fundi, suggested tumor of the cerebellum as the correct diagnosis; both of these diagnoses were, therefore, entertained. The patient was referred to the wards of the Hospital and remained under observation from November 8 to November 16, 1915. The symptoms and signs already noted did not change and the patient was discharged from the hospital as a case of Friedreich's ataxia. There was no definite evidence, except the gait, to justify a diagnosis of cerebellar tumor.

The patient was seen again in the Out-Patient Department on December 11, 1915. Considerable improvement had taken place at that time. By March 25, 1916 the patient was practically well: his gait was normal; there was no ataxia of the arms or legs; the Romberg sign had disappeared; the knee jerks and ankle jerks had become equal and active. Some surprise was expressed at that time by the examiner in regard to the original diagnosis.

The patient was seen again in the Out-Patient Department fourteen years later, November 19, 1929. There were no indications of any marked deviation from normal health and the patient had, between the years 1917 and 1929, served for a period of over three years in the U. S. Army. His appearance in 1929 gave no indication of Friedreich's ataxia inasmuch as his gait was normal, no ataxia was present, and the reflexes were equal and active.

In 1915 this patient presented a picture consistent with that seen in Friedreich's ataxia in that he had a well marked ataxic gait, the deep reflexes were absent, and he was about the correct age. There was, however, no family history of this rare disease, the onset had been relatively sudden; speech defects, nystagmus or pes equino-varus were lacking. These latter points, however, were set aside by four or five observers in favor of the diagnosis of Friedreich's ataxia. In view of what we know now, having seen this patient fourteen years later, we would conclude that the correct diagnosis in 1915 should have been peripheral neuritis, probably of the infectious variety, with very little pain and no definite paresthesia or anesthesia. The neuritis might possibly have been due to one of the heavy metals such as lead, but this does not seem to be likely in view of the fact that recovery, once started, proceeded rapidly. It must be said, however, that the patient took potassium iodide by mouth for a number of months and this therapy may have shortened his illness. One cannot be certain, at this late date, of the cause of the neuritis.

Peripheral neuritis is a disease of many varieties. It should always be considered in any patient who loses strength, becomes ataxic, and has loss of deep reflexes, within a short period of time, even if there be no pain connected with the disease.

HENRY R. VIETS, M.D.

Boston, Mass.
November 25, 1929.

COMMENTS ON THE MEDICAL PROBLEMS INHERENT IN ALLEGED MIRACULOUS CURES

In order that the sensational demonstrations of faith healing in Malden may not become on a vast scale a real menace to public health—if not to faith itself—it would seem desirable to acquaint readers with the following easily confirmed facts:

1. Many of the cases reported "cured" are, from their very nature, such that the reports of the patients or the bystander are of no scientific value whatsoever, and often tend to mislead the public into the belief that a large number of miracles are taking place, when, in reality, nothing has been proven.

2. Possibly fifty per cent. of the more sensational cases contain the hysterical element and furnish the basis for much more in the way of mental suggestion and imitation. The hysterical patient invariably takes the center of the stage and gets most of the limelight by the most theatrical displays of his own dramatic conceptions of more or less imaginary ailments. They frequently fool skilful physicians. Is it any wonder that they delude the uninformed?

3. The next prominent group are "cripples," and must be divided into at least three classes:

(a) Tubercular. Here the joint may be painless. Splints are worn to fix the joint which otherwise would be injured by normal movements. Only an expert can recognize these lesions or tell when they are cured. Ability to walk without the splint is no proof of cure, but such activity may lead to disaster, in place of permanent recovery in months to come.

(b) Infantile paralysis. These cases have lost the use of muscles which later degenerate, causing serious deformities. Casts and supports are worn until danger of this deformity has passed. Here again nothing is proved by the ability to walk without props.

(c) Chronic arthritis from many causes. Falls, sprains, fractures, dislocations and previous attacks of acute arthritis which have already healed. These, almost without exception, need limbering up, and if, under emotional excitement, patients can be induced to do a little of this for themselves, instead of going to a masseur, so much the better. Where are the miracles?

What is there left to consider? We may mention advanced cancer, obscure sex diseases, and the acute infections such as diphtheria, smallpox, typhoid fever—surely not the cases to gather about a shrine, kissing a chalice and bathing their sores from a common receptacle.

We read of pilgrims applying the soft, trampled mud over their faces, arms and legs, and to the eyes of infants! If so, are these exhibitions an aid to faith? I doubt it. Thinking people today stand aghast, not knowing what to say, but most of them are ruling in their hearts that if God cannot heal bodies by methods more sanitary and less revolting—well, they will trust to a clean hospital, up-to-date nurses, and doctors with a reputation at least for intelligence.

WILLIAM W. HARVEY, M.D.

RECENT DEATH

FRENCH—DR. CHARLES LINDOL FRENCH, of Clinton, died at St. Petersburg, Florida, November 18, 1929, aged 83.

Dr. French was a native of Vermont where he received his early education, later graduating from Columbia University College of Physicians and Surgeons in 1869. He practiced for a short time in Vermont and then moved to Clinton, Massachusetts. He joined the Massachusetts Medical Society in 1887.

He went to Florida in October planning to spend the winter; suffered a broken hip from a fall in his apartment early in November and died of pneumonia.

In addition to a large practice throughout northern Worcester County, he was prominent in the founding and development of the Clinton Hospital, serving on its medical staff and the board of trustees until his death.

His interest in civic matters led to service on many local boards and committees. He enjoyed to an unusual degree the regard and affection of the community.

As a physician he exhibited loyalty and tender regard for his patients and until within a few years found more enjoyment in the practice of medicine than in vacations or forms of relaxation generally adopted by men of prominence.

His wife, who carried many of the detailed responsibilities of a doctor's life, died several years ago.

He is survived by a son, Hon. Harold L. French of Clinton, Massachusetts, and Boston, and a daughter, Mrs. George C. Graham of Watertown, Massachusetts.

OBITUARY

RESOLUTIONS PASSED BY THE TRUSTEES OF THE BOSTON CITY HOSPITAL ON THE DEATH OF DR. EDWARD N. LIBBY

The Trustees of the Boston City Hospital wish to record their sense of great personal loss in the death of Dr. Edward N. Libby. For thirty years he gave distinguished service to the Hospital, whose welfare was one of his chief interests. An exceptional teacher, he inspired his students with the best traditions of his profession; an able diagnostician, he was widely sought as a consultant, but he was no less eminent in representing the highest type of the family physician. As their physician and friend, he gave with self-sacrificing devotion the same full measure of his talents to all his patients, whether in private practice or in the wards of the Hospital. His modesty, generosity, sanity and wisdom, made him the trusted adviser of his colleagues and the Trustees. The Trustees send their heartfelt sympathy to his family, in whose sorrow they share.

It was voted that a copy of the foregoing be sent to the widow of Dr. Libby, and to the NEW ENGLAND JOURNAL OF MEDICINE.

NEWS ITEMS

RUTLAND HOSPITAL NEEDS MORE BEDS—It has been reported by General Frank T. Hines, Director of the Veterans' Bureau, that more beds are needed at the Government Hospital at Rutland. Some of the veterans afflicted with tuberculosis have been sent to the Chelsea Hospital, to meet the present demand. Chelsea Hospital is not a proper location for treatment of tuberculosis. From thirty to forty beds are needed and immediate action to relieve the situation is urged.

HARVARD MEDICAL SCHOOL NEWS—The following resignations and appointments were made public by the Dean's Office on November 19, 1929.

New appointments: One year from September 1, 1929:

Austin Walter Cheever, M.D., Assistant in Dermatology and Syphilology.

LeRoy Dryden Fothergill, M.D., Assistant in Bacteriology.

Harry Aaron Derow, M.D., Teaching Fellow in Medicine.

Edward Tenney Casswell Spooner, M.R.C.S., L.R.C.P., Research Fellow in Bacteriology.

Moses Myer Suzman, M.D., Research Fellow in Medicine.

New appointment: From September 1, 1929 to July 1, 1930:

Andrea Svedberg, Med.Lic., Research Fellow in Biological Chemistry.

Resignations:

William Lorenzo Moss, M.D., Assistant Professor of Bacteriology and Immunology, effective September 1, 1929.

Frank Brostrom, M.D., effective November 15, 1929.

RADIUM FOR THE ROYAL VICTORIA HOSPITAL—Ten prominent Montreal residents have donated $25,000 worth of radium to the Royal Victoria Hospital for the treatment of cancer. An equal donation will be made to the Montreal General Hospital.

THE APPOINTMENT OF DR. MAY AND DR. MYERSON TO MAKE A STUDY OF A CRIMINAL—Dr. May and Dr. Myerson have been appointed by Dr. A. Warren Stearns, State Commissioner of Correction, to determine the mental responsibility of Charles Trippi, Jr., who killed Frederick Pfluger, a State Prison guard, while trying to escape a year ago. Dr. May and Dr. Myerson have reported that Trippi is sane.

MASSACHUSETTS LAGS BEHIND IN SOME PHASES OF PUBLIC HEALTH WORK—At a recent meeting of the Women's Republican Club Dr. George H. Bigelow, State Commissioner of Public Health, informed those present that Massachusetts is behind some other states in trained public health service in its rural communities. Other speakers were: Dr. W. S. Draper, assistant surgeon-general of the United States Public Health Service; Dr. Paul B. Brooks, deputy health commissioner of New York; and Dr. Wilson G. Smillie, professor of public health administration at the Harvard School of Public Health.

ANOTHER EXPLOSION IN AN X-RAY DEPARTMENT—Reports of an explosion in the X-ray department of the University of California Hospital, which started a fire resulting in several thousand dollars of damage, have been published.

Fortunately no lives were lost. The Massachusetts authorities are requiring especial care of films and apparatus with the hope that similar troubles may be avoided.

APPOINTMENT OF DR. FRANCIS D. DONOGHUE—Mayor Nichols has appointed Dr. Francis D. Donoghue to the position of Commissioner of school buildings of the City of Boston.

The board will take over the functions of the school home commission as provided under the recently accepted referenda.

SPINAL ANAESTHETIC IN CAESAREAN SECTION—The papers report that Dr. Francis D. Donoghue performed a caesarean section on a patient in Whitman in 1912 which is claimed to have been the first application of spinal anaesthesia in caesarean operations. This may be of sufficient interest to lead to further reference to this type of work.

NOTICES

WARNING TO PHYSICIANS

Recently there has been active among physicians in New York and Boston, an impostor, who on two occasions has represented himself to be the son of Dr. Otis B. Wight of Portland, Oregon. The impostor is about five feet, eight inches in height, with dark hair and eyes so dark that except in a good light his pupils cannot be distinguished. His face is rather long and narrow with well-developed nose and chin, lips slightly full, skin pale and fairly clear, well dressed, wearing a fraternity pin prominently (not identified but claimed by the wearer to be Sigma Chi); pleasant manners and address. When last seen he had on a light gray felt hat, light brownish gray top coat, dark blue suit, tan shoes and rubbers. He claimed to have been at Johns Hopkins Medical School two years and to be now at Western Reserve University in the fourth year of medical school. His name and story will probably be different when next heard from but a description of him has been given to the Boston police department. Dr. Wight is interested in his apprehension.

UNITED STATES CIVIL SERVICE EXAMINATIONS

The United States Civil Service Commission announces the following open competitive examinations:

Senior Medical Officer (Internal Medicine), $4,600 a Year

Junior Medical Officer (Interne), $2,000 a Year

Applications for senior medical officer (internal medicine) and junior medical officer (interne) must be on file with the Secretary of the Fourth U. S. Civil Service District, Washington, D. C., not later than December 26.

The examinations are to fill vacancies in Saint Elizabeth's Hospital, Washington, D. C., and vacancies occurring in positions requiring similar qualifications.

REMOVAL

Dr. Bancroft Cheever Wheeler has announced the opening of his office at 27 Elm Street, Worcester, Massachusetts.

RHODE ISLAND REPRESENTATIVES AT THE CONFERENCE OF SECRETARIES AND EDITORS IN CHICAGO

Dr. F. N. Brown. Editor of the *Journal of the Rhode Island Medical Society*, and Dr. J. W. Leech, Secretary of the Rhode Island State Medical Society, attended this conference.

These names should have been included in the earlier reference to this meeting in the issue of November 21, page 1068.

DIVISION OF THE BLIND OF THE DEPARTMENT OF EDUCATION

Information of the work of this Division can be obtained on application to Robert I. Bramhall, Director.

Facts relating to blind men should be known by all public spirited citizens. A circular of information is available for all who apply for it.

REPORTS AND NOTICES OF MEETINGS

NEW HAVEN MEDICAL ASSOCIATION

(Continued from page 1071, issue of November 21.)

Dr. Joseph C. Aub, Assistant Professor of Medicine at Harvard University, discussed "The Influence of Internal Secretions on Metabolism." Reference was also made to the effects of inorganic salts, especially calcium, on metabolism. Administration of parathormone, a parathyroid hormone, to patients with low blood calcium and tetany removes the symptoms by raising the blood calcium. There is also an increased calcium excretion. This parathormone treatment is not continually effective, for an immunity to such treatment develops in a few weeks.

Exophthalmic goitre patients excrete large amounts of calcium but thyroidectomy reduces this excretion. If a normal or myxedematous person takes thyroid extract, an increased calcium excretion results. Thyroid speeds up calcium metabolism in a way that is out of proportion to its effect on the metabolism of organic materials.

A high acid diet causes an increased calcium excretion. Most of this calcium excretion occurs through the urine, and practically none is found in the feces. A high acid diet differs from parathormone in not raising the blood calcium.

That bone is the storehouse for calcium may be shown in several ways. X-ray plates show that myxedematous patients have thickened bone. In hyperthyroidism there is usually found a thinning of the bone. A more direct proof was offered by experiments which showed an increased trabeculation at the epiphyses of bones of experimental animals that had been fed a high calcium diet. On a low calcium diet these trabeculations diminished. Special staining of epiphyseal sections showed that calcium is deposited in the trabeculae.

Dr. Aub is well known as an authority on lead poisoning. On the day following his above lecture to the New Haven Medical Association, Dr. Aub discussed lead poisoning at the Yale Medical School.

Patients with lead poisoning have a peculiar pallor which is characteristic. Practically all patients who have lead poisoning have poor teeth. Caries and marked pyorrhea is usually seen. With pyorrhea there is usually hydrogen sulphide on the gums. Lead sulphide then forms around the arterioles of the gums when lead is absorbed. In examining a lead line on the gums, the gums should first be well cleaned. A lens is used in the examination and a punctate dotting is seen. The black line on the gums may also be caused by bismuth and mercury. Iron may sometimes give this black line. A false "lead line" is sometimes seen in negroes. The black line is pathognomonic of lead absorption and not necessarily of lead poisoning.

Abdominal symptoms in lead poisoning must be differentiated from acute abdominal conditions such as—intussusception, gastric ulcer and acute appendicitis. Acute abdominal conditions may complicate a case of lead poisoning. With lead colic there is practically never any rise in temperature. The white blood cell count as a rule is not increased. A patient with lead colic usually has constipation for two days before diarrhea occurs. Much aid for differential diagnosis can usually be gotten by asking the patient where the pain is. The patient usually touches the whole of the abdomen with his hand, in lead colic. In a case of gastric ulcer the patient usually points with his finger to one spot in the epigastrium. In appendicitis the pain is usually localized at McBurney's point. The pain in lead colic is due to a contraction of smooth muscle. With lead colic there is no true abdominal spasm, localized in one spot, as is usually found in cases of gastric ulcer or appendicitis. With lead colic there may be spasms of the abdominal muscles over the area of intestinal spasms, so the abdominal spasms may be found first in one area and then in another. Some patients with lead colic seem to have a tendency to gastric ulcers. In some complicated cases of lead poisoning fever and leucocytosis may be found. Patients with lead colic are often found to have "scaphoid abdomens."

Cases of lead poisoning must be differentiated from peripheral nerve lesions.

Patients with lead poisoning have practically no peripheral sensory disorder but usually have a motor paralysis of the muscles which are used most. The muscles which are relatively weak, e.g., the extensors of the forearm, are usually affected. Some hold that the selection of the extensor muscles has to do with chronaxia. The reason is not clear.

Usually the extensor muscles of the first, middle and ring fingers are first involved. Then the wrist becomes involved. The fingers become weak and then paralyzed. In cases in which paralysis is not yet reached the fingers may be held up and then regardless of how much the patient tries he cannot keep them from gradually dropping down.

It is very important to treat "lead poisoning patients" before paralysis occurs. With weakened muscles the patient can be cured in six weeks. With paralyzed muscles at least six months is needed before cure.

Several tests are had to demonstrate the peripheral neuritis of the motor nerves. When the patient holds his arm out, the wrist as well as the fingers will soon show tremor and then gradually drop. Both fingers and wrist are in a flexed position. This is not a true paralysis but a weakness of the extensor muscles. Weakness of muscles may also be demon-

strated when the patient squeezes the examiner's hand. When the patient tries to button his coat the stronger flexors may be seen to gain control. Another test to demonstrate weakness of the extensors: When the arm of the patient is pulled from a flexed position, strength is demonstrated by the patient, but when the arm is pushed back there is little resistance by the extensor muscles. In some cases an atrophy of the deltoid and scapular muscles is had so that use of the arm is decreased. If the process continued without treatment there would eventually be a complete flaccid paralysis with no sensory change. At times sensitiveness may be elicited in the tendon sheaths. This is probably due to a stretching of the tendon sheath.

Jaundice is a symptom which may be marked in patients with lead poisoning. This jaundice is due to a peripheral destruction of red blood cells. The peripheral red blood cells at first become fragile and then break. Anemia is also found.

In the history of lead poison patients the occupation of the one involved is of utmost importance. An exact knowledge of the specific kind of work the patient does should be had, because in cases of exposure the patient would be spending about one-third of each day at such work.

In regard to the course of lead poisoning:—Lead may get into the body by swallowing it or through the mucous membrane of the respiratory tract. Lead is ten times more toxic when absorbed through the respiratory tract than when absorbed through the gastro-intestinal tract. When lead is absorbed by the gastro-intestinal tract much of it goes to the liver where it is extracted by the Kupffer cells. From the liver it is excreted into the bowel. However a slight amount of lead reaches the bone in these cases and is deposited there. However, when lead is absorbed by the respiratory tract it soon gets to the general circulation and then to the susceptible tissues—smooth muscles, nerves and bones. So this accounts for lead being ten times more toxic when absorbed by the respiratory tract than when absorbed by the gastro-intestinal tract. So in industry lead dust and fumes should be concerned more than lead swallowed. Lead soon gets to the blood and causes anemia. On account of the anemia many reticulocytes appear. Many of these young red blood cells become stippled. In stippling small black dots are seen in the reticulocytes. Stippling is also found in leukemia, pernicious anemia, and sometimes in pneumonia of children. However in lead poisoning there are from one hundred to two hundred stippled red blood cells per million. Anemia and stippling of red blood cells are the first signs of lead intoxication. Lead is soon carried by means of the circulation to bone where it is stored. If there was no storage place for lead, it would be one of the greatest poisons to deal with. Lead is deposited in bone as a triphosphate, insoluble salt. When lead is stored in bones there is no injury done. It is only when in circulation that damage is done. However, lead may be liberated from bone when calcium is liberated to neutralize acids. This is especially seen in chronic alcoholics—carbohydrate is broken down; protein of the body is broken down; phosphates and sulphates are liberated; calcium comes out of bone and with the calcium, lead. So lead stored in bone over a long period of exposure may not cause symptoms until an acid condition persists.

Besides affecting blood and smooth muscle lead also affects striated muscle to a slight extent, and nervous tissue. Ten per cent. of lead intoxication cases develop paralysis of the arm. Five per cent. develop encephalopathy which is characterized by convulsions, hallucinations and delirium. On lumbar puncture there is found an increase in spinal fluid pressure.

In treating lead intoxication patients it must be remembered that lead is excreted mainly in the feces. Only a slight trace is excreted in the urine. So the patient must have at least one movement a day.

On the first day the patient is seen, he is usually suffering from the very severe colic pain. Most patients flex the femurs on the abdomen to get relief. The colic pain is caused by a contraction of a part of the intestine with the peristaltic waves continuing to come down from above. When the contraction is released there is no more pain. First an enema should be given. Then atropine and magnesium sulphate may be given. Next, ten or 15 c. c. of 5 per cent. calcium chloride is injected intravenously. This calcium should be injected very slowly. At least five minutes should be consumed during the injection for calcium tends to depress the heart. If calcium is not injected into the vein it causes a slough. After seven c. c. are given the patients usually "flush up" due to dilation of the peripheral blood vessels by the calcium chloride. The patients feel hot. Giving 2 c. c. more of calcium chloride is of no great use unless it makes the patients feel hot. Soon the patients feel tired and usually go to sleep. When quiet give magnesium sulphate to eliminate feces with lead. Keep bowels open. If patients have pain in five days examine for gastric ulcer. The diet should next be improved. Give a high calcium diet—one quart of milk a day, green vegetables, and eggs. Calcium lactate may also be given in the diet. Keep calcium flowing toward the bones for three days. After three days, draw lead from the bone. So low calcium diet is now fed. To get rid of more calcium give an acid diet slowly to draw calcium and lead from bone. The best way is to give doses of ammonium chloride. The ammonium goes to form urea, leaving the chloride free. Some give capsules of calcium chloride at meals. Symptoms of nausea, loss of appetite and dullness may occur. Parathormone pulls calcium and lead out of bone. It does this very well but parathormone is a very active principle and must be controlled by blood calcium determinations. Some say that lead should not necessarily be pulled out of bone. However, with sickness or an acid state the lead would later come out. When bone is depleted the trabeculae at epiphyses are attacked and practically none of the solid bone is used. So lead has a tendency to deposit in the trabeculae. After lead is pulled from bones and excreted, a calcium diet is given.

(To be continued)

MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE

The Massachusetts Society for Mental Hygiene held its Annual Meeting on November 13 at the Twentieth Century Club, Boston. Meyer Bloomfield, Consultant on Industrial Relations in New York City, addressed the group on the subject of "Preventive Management." He discussed the philosophy of management which is changing from the older conception of patching up difficulties and of remedying faults to that of prevention. As examples of this, he mentioned plans for preventing unemployment and for utilizing aged employees in industry; he also spoke of the importance of mental factors as causes of industrial unrest and recommended that methods should be used to minimize this source of maladjustment; and finally, he stated that the worker should take upon himself the obligation to give his most efficient work in return for his wages, and that the worker as well as the executive should look to scientific and preventive management for the future welfare of industry.

Dr. A. Warren Stearns, Massachusetts Commissioner of the Department of Correction, was the second speaker, his subject being "Psychiatry in Penal Administration." Dr. Stearns attempted to reconcile the practical points involved in applying new sociological and psychiatric theories to penal practice. It is his opinion that there will not be enough psychiatrists to fill the ranks in prison administration and that therefore a new class of socially trained individuals should be developed to carry on the work. Dr. Stearns believes that although psychiatry's usefulness to penology has been overstressed, it has an important contribution to make in that it provides a scientific and practical method of approach to the problems of penology.

The following officers and members of the Executive Committee were elected: President, Dr. C. Macfie Campbell; Vice President, Hon. Herbert C. Parsons; Secretary, Dr. Charles E. Thompson; Treasurer, Romney Spring, Esq., Assistant Treasurer, Dr. Donald Gregg; Executive Committee: Dr. E. Stanley Abbott, Hon. Frederick P. Cabot, Dr. Ransom A. Greene, Dr. William Healy, Mrs. Robert F. Herrick, Mr. Henry S. Dennison, and Miss Mary Lee Ware.

THE NEW ENGLAND OBSTETRICAL AND GYNECOLOGICAL SOCIETY

The first meeting of the New England Obstetrical and Gynecological Society was held in Boston, November 20, 1929 the day being given over to clinics at the Brookline Free Hospital for Women, Boston City Hospital Gynecological and Obstetrical Service, St. Elizabeth's Hospital, Carney Hospital, Homeopathic Hospital, Boston Lying-In Hospital, Booth Hospital and Huntington Hospital.

The social attractions were staged in the evening at the University Club where, after the business meeting an elaborate banquet was served followed by an address by Dr. Mongan, the President, in which a history of the movements leading to the formation of the Society was given. In response to the first toast Dr. R. B. Greenough, President of the Massachusetts Medical Society, brought the greetings of the Society to this daughter organization, for such it seemed to be because of the influence of the Section of the Massachusetts Medical Society in creating interest in this larger body. He urged especial attention to some of the problems of cancer which will have to be studied by members of this Society. He was followed by Dr. Morris Fishbein, Editor of the *Journal of the American Medical Association*, who entertained the Society and guests by an address dealing with the various types of medical practice in which humor, pathos and satire were mingled with telling effect. This subject gave opportunity for a display of the unusual power of this gifted editor, who is regarded as one of the brilliant public speakers among physicians. His address is not available for publication because it has been reserved for especial occasions, but if the opportunity offers any who have not been privileged to hear it would be amply repaid for any trouble incident to attending a meeting at which it will be repeated.

This addition to the Medical Societies of New England promises to be of great importance because of its influence in dealing with the vital matters incident to pregnancy and medical education.

This JOURNAL has been honored in being selected as the official organ of this Society. We anticipate extending the usefulness of the JOURNAL through the publication of the proceedings of the New England Obstetrical and Gynecological Society.

A PUBLIC MEETING AT THE BETH ISRAEL HOSPITAL TO HEAR DR. FISHBEIN DISCUSS THE COST OF MEDICAL CARE

On November 21, 1929 Dr. Charles F. Wilinsky, Director of the Beth Israel Hospital, invited doctors and laymen to meet in the Auditorium of the Beth Israel Hospital to meet Dr. Fishbein and listen to an address on the Cost of Medical Care.

Through his extensive experience and contact with physicians and organizations interested in the economic problems of Medical Care, Dr. Fishbein is especially qualified to discuss this subject.

In the brief time at his disposal because of the facility of expression of the working of his active mind the economic changes in the social life of the nation were set forth clearly thereby showing that many factors enter into the problems many of which are concerned with the elaborate equipment of experts and hospitals and the greater expenses incident to the standards of living of the present time.

In addition to these the unnecessary luxuries contribute no small part to the dissipation of the funds of people in moderate circumstances who in consequence find difficulty in meeting the unexpected expenses of illness.

This was a very instructive address, the arguments being emphasized by telling illustrations.

Here again the subject enabled this gifted speaker to deliver an address which was of educational value in addition to being of absorbing interest.

HARVARD MEDICAL SOCIETY

The Harvard Medical Society held a meeting in the Peter Bent Brigham Hospital Amphitheatre on Tuesday, November 12, 1929, at 8:15 o'clock. After the presentation of cases Dr. S. B. Wohlbach introduced Dr. Cecil K. Drinker, Professor of Physiology at the Harvard School of Public Health, who spoke on "Lymphatics and Lymphatic Drainage."

The first case was presented by Dr. Light of the surgical service. The patient was a 44 year old Belgian married housewife who entered the hospital a month ago complaining of persistent pain in the left lower abdominal quadrant accompanied by vomiting for six weeks. Seven years before she had had an attack of cholecystitis which was repeated last July when she had her gall bladder removed. She remained well for a month and then pain in the left lower quadrant with vomiting occurred, which attacks recurred at intervals of a few days. Physical examination on entry showed a markedly emaciated and dehydrated woman with a soft abdomen containing a mass in the left upper quadrant, diagnosed as a movable kidney, and a small mass in the left lower quadrant which later disappeared. X-ray showed a normal colon, but barium by mouth showed a partial intestinal obstruction. Operation revealed an annular constricting lesion with enlarged mesenteric lymph nodes affecting the small bowel. A side to side anastomosis was done and closed without drainage with a view to letting the patient recover her strength before excision was attempted. At the second operation a large mass was felt in the duodenal mesentery completely overshadowing the tumor of the intestines. Biopsy confirmed the diagnosis of a rapidly growing tumor of the lymph nodes and the wound was closed. The pathologist reported that the behavior of the tumor was more like a carcinoma than a lymphoma.

The second case was presented by Dr. Harrison. The patient was a single white English-man who came to America six years ago. He came into the hospital four days before complaining of a rash and painful joints. His history revealed that he had measles at three, and scarlet fever at seven. His present illness began 14 years before entry. In 1915 he had an attack of joint pains in both ankles, lasting 2-3 days. He had similar attacks in 1919, 1923. In 1925 the pain came on in his shoulders. In 1926 his tonsils were removed. In 1928 his knees became swollen and painful. With each attack he had had a sore throat and with one or two he went to bed. Three weeks ago he developed a sore throat, anorexia, and a "run-down" feeling. Two weeks ago he began to notice pains and stiffness in his left ankle and right hip, which pains spread to other joints. A rash developed on his back which spread to abdomen, chest, and thighs. Physical examination on entry showed a man in no real pain with bad teeth, no glandular hypertrophy, and generally stiff joints. The rash was erythematous in nature, blanched on pressure, the edges were elevated, it was not painful or itchy. Reflexes, heart, and lungs were essentially normal. Laboratory work revealed a normal clotting and bleeding time and a negative tourniquet test. The case was presented as an example of that group of purpuras where the pathology is in the capillaries. This type has been called anaphylactoid because of its resemblance to manifestations of serum sickness which is an anaphylactic reaction.

Dr. Drinker first pointed out that the lymphatic function is at the basis of body repair and of limitation of disease processes. In studying the comparative anatomy of lymphatics, it is found that no such channels are present lower in the scale than fishes. Below these the transfer of water, proteins, and salts is accomplished without two sets of vessels. In the frog there is a good lymph system and its ramifications are just as numerous and complex as the blood vessels. The lymph leaves and returns to the blood about fifty times every twenty-four hours. Thus, in the frog the lymph system is probably circulatory in nature. In mammals the flow is similar in the main, but not nearly so fast. Sabin has shown that lymphatics are derived from the veins.

In studying the composition of lymph it is found to contain 4% protein which is albumen, globulin, fibrinogen and all the other elements necessary for clotting. The osmotic pressure of blood protein is between twenty and forty mm. of mercury; variations in this pressure cause fluid exchange between blood and the tissue spaces. If the capillaries are impermeable to protein, as some believe, where does the lymph protein come from? Dr. Drinker believes that it must come from the capillaries. If protein is injected subcutaneously, it has been shown to be absorbed into the lymph first and then into the blood. Two hours suffice for this absorption.

It is known that water, salt, and traces of protein leave the blood, while only water and salt return. Thus, the lymphatics must remove or clear out protein materials from the tissue spaces. Dr. Drinker suggested that perhaps a function of the lymphatics is to maintain a constant protein concentration in the tissue spaces. Applying this theory to practice, if a tissue is cultured on a high protein medium, growth results. Thus, when a body wound occurs, plasma and lymph accumulate forming a high protein medium for connective tissue growth. One may conclude that the minimum of cicatrix depends on efficient lymph drainage. In support of this, Billroth has shown that in a heavy cicatrix lymphatics are very scarce and grow slowly.

In conditions such as filariasis or other lesions causing lymph blocking, the limbs and parts are not permanently large because of edema, but because of fibrosis. There is more fibrosis in filariasis because there is usually a concomitant infection superimposed on the parasitic infection causing hyperemia and better conditions for fibrosis resulting. Thus, those patients with a good circulation get more fibrosis. This explains the little fibrosis in cardiac edemas.

Dr. Drinker concluded by pointing out exceptions to his hypothesis. Why progressive fibrosis does not occur in the spleen, liver, and lymph nodes he could not explain. Dr. Cannon suggested that stasis might be a necessary factor in producing fibrosis, the lymph and blood moving fairly fast in those organs where one would expect fibrosis due to this cause.

The meeting was closed with interesting discussion by Drs. Cannon, Homans, and Wohlbach.

THE DEDICATION OF THE HEALTH UNIT IN THE CHARLESTOWN DISTRICT

A large and enthusiastic body of doctors and laymen gathered at the Fifth Health Unit, High and Elm Streets, Charlestown, November 22, to witness and participate in the dedicatory exercises of this last addition to the group of buildings which has been and is still to be erected under the provisions of the George Robert White Fund.

After an inspection of this latest conception of Mr. Charles A. Coolidge of the firm of Coolidge, Shepley, Bulfinch and Abbott by doctors, public health officials and the public, Mr. George E. Phelan, Manager of the White Fund, called the meeting to order and after extending a cordial welcome to those present explained the progress under way by the trustees of the fund in creating the five units now scattered throughout Boston with one other now in process of building. He called upon the Reverend Henry Lyons, Pastor of St. Catherine's Church, Charlestown, to pronounce the invocation.

Mr. Coolidge then presented the keys of the building to the Hon. Herbert Parker, a member of the Board of Trustees and President of the Boston Bar Association, who gracefully responded for his colleagues and, acting under the provisions of the will of the founder of the fund, transferred the unit to the city of Boston as represented by his honor, Malcolm E. Nichols, who extolled the city of Boston in its capacity to carry on the great work made possible by the creator of the fund and placed the administration of the work in the hands of the Health Department and Dr. Francis X. Mahoney. Dr. Mahoney explained the co-ördinate interest of other bodies and predicted that within a few years the public health program of the city will be so strengthened by these units that the results will be commensurate with the expectations of the wise and liberal minded founder of the Fund.

Dr. John F. O'Brien, past president of the Charlestown Medical Society, gave a brief history of the public health activities of that section and in behalf of the physicians of Charlestown welcomed this addition to the medical service in this district and pledged the co-öperation of the physicians located therein. The Hon. Thomas H. Green, member of the City Council, spoke at length of the interest of the citizens of Boston and its officials in maintaining the highest type of civic virtues and the Hon. Charles S. Sullivan, Justice of the Municipal Court, extended the greetings of the bar.

Although not on the printed program the presence of Mayor-elect James M. Curley made his introduction especially fitting because the first unit was established and dedicated during his earlier administration and the public health policies of the city had always been profoundly alluring to him. He paid tender tribute to the memory of George Robert White and to Mrs. Bradbury, sister of Mr. White, who had been an inspiring adviser to those who are responsible for the administration of the trust, and to the clergy of the district who had taken deep interest in the spiritual and material welfare of the citizens of that community.

The dedicatory address was delivered by Professor C.-E. A. Winslow, Dr. P.H. and Professor of Public Health at the Yale School of Medicine. In opening his remarks Dr. Winslow expressed his pleasure at being present both because of his interest in the work and because of his early life in this city and its educational institutions. In speaking of the significance of health units Dr. Winslow reminded the audience of the history of the development of human ideals as expressed first through the symbols of spiritual life exhibited in the building of churches, second by educational institutions in the development of intellectual growth and third in that symbol of community life which is centered in the recognition of the needs of the physical being under the designation of health.

He recognized in Boston and Massachusetts leadership in health activities especially in the programs dealing with tuberculosis, cancer and mental hygiene, and in this creation of health units Boston leads the world. In emphasizing the agencies which have been especially potent in these plans he placed great credit upon the enthusiastic, wise and energetic work done by Dr. Charles A. Wilinsky who has the great credit of directing the attention of Mayor Curley and through him the trustees of the fund to the great possibilities in promoting the health of the people by means of well equipped and properly conducted Health Units. The united testimony of the speakers gave to Dr. Wilinsky the high honor of being the first to conceive the possibilities of these units and to demonstrate their efficiency as the director of the earlier ones.

MASSACHUSETTS PSYCHIATRIC SOCIETY

Professor Eugen Bleuler, formerly director of the Psychiatric Clinic at Zurich, Switzerland, will be the guest speaker of the Society at its next meeting. This meeting will be held in the John Ware Hall of the Boston Medical Library, 8 The Fenway, on the evening of Saturday, December 7, at 8:00 o'clock. His subject will be "Physiologic and Psychic Symptoms in Schizophrenia."

WINFRED OVERHOLSER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

1929-1930—Massachusetts Dietetic Association. Complete schedule appears on page 852, issue of October 24.

December 2-6—The Radiological Society of North America. Complete notice appears on page 149, issue of July 18.

December 7—Massachusetts Psychiatric Society. Complete notice appears above.

December 16-17—The American Pharmaceutical Association. Complete notice appears on page 1019, issue of November 14.

February 10-14, 1930—American College of Physicians. Complete notice appears on page 853, issue of October 24.

March-October, 1930—International Medical Postgraduate Courses in Berlin. Complete notice appears on page 853, issue of October 24.

May 5-10, 1930—The International Congress on Mental Hygiene. Detailed notice appears on page 853, issue of October 24.

September 4-8, 1930—Fifth International Congress on Physiotherapy. Complete notice appears on page 906, issue of October 31.

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

January 8, 1930—Wednesday—Semi-annual meeting at Centre Church Vestries, Main Street, corner of Vestry Street, opposite City Hall, Haverhill, (Telephone 548) at 12:30 sharp.

May 1—Thursday—Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

May 7—Wednesday—Annual Meeting.

J. FORREST BURNHAM, Secretary.

Essex South District Medical Society

Wednesday, December 4, 1929—Salem Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Howard B. Sprague, of Massachusetts General Hospital Heart Clinic. Subject: "Recent Progress in the Diagnosis and Treatment of Heart Disease." General Discussion from the floor.

Wednesday, January 8, 1930—At the Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Harry H. Germain, Boston. Subject: "Some Aspects of Cancer of the Colon." General Discussion from the floor.

Wednesday, February 5, 1930—Council Meeting, Boston.

Wednesday, February 12, 1930—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speakers: Dr. Winfred Overholser. Subject: "Criminology and Psychiatry." Mr. Clark, District Attorney for Essex County, who will speak on the legal aspect.

Wednesday, March 5, 1930—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. R. S. Titus, Boston. Subject: "Some Aspects of Caesarean Section," illustrated with Moving Pictures. General Discussion from the floor.

Wednesday, April 2, 1930—Hotel Hawthorne, Salem. Dinner at 7 P. M. Speaker: Dr. Timothy Leary, Boston. Subject: "Diagnostic and Therapeutic Pitfalls in Intracranial Pyogenic Infections."

Thursday, May 1, 1930—Censors meet for Examination of Candidates at Salem Hospital at 3:30 P. M.

Tuesday, May 13, 1930—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. C. Macfie Campbell, Director of Boston Psychopathic Hospital. Subject to be announced later. Ladies invited. Dancing.

R. E. STONE, M.D., Secretary.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held at the Weldon Hotel, Greenfield, Mass., on the second Tuesdays of January, March and May, at 11 A. M.

CHARLES MOLINE, Secretary.

Hampshire District Medical Society

The meetings of Hampshire District will occur at 11 A. M. the second Wednesdays of December, February, and April and will be held at the Dickinson Hospital, Northampton. The annual meeting will be held in May.

LUTHER O. WHITMAN, M.D., Secretary.

Middlesex East District Medical Society

January 15—At Boston, Harvard Club.

March 12—At Melrose.

May 13—At Unicorn Country Club, Stoneham.

ALLAN R. CUNNINGHAM, Secretary.

Middlesex South District Medical Society

Winter meetings—jointly with Suffolk District.

April 16, 1930—Annual meeting. Commander Hotel, Cambridge. Program to be announced.

May, 1930—Censors' meeting.

ALEXANDER A. LEVI, Secretary.

Norfolk District Medical Society

January 27, 1930—Roxbury Masonic Temple. "Recent Advances in Diagnosis and Treatment of Syphilis." The speakers will be Drs. C. Morton Smith and Austin W. Cheever.

February 24, 1930—Roxbury Masonic Temple, 8:00. Dr. Edwin H. Place has been invited to read a paper and has been given carte blanche in the selection of his subject.

March 24, 1930—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

May 6, 1930—Annual Meeting. Program to be announced.

The Censors meet for the examination of candidates, May 1, 1930, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M.

Applications must be in the hands of the Secretary at least one week previous to date of examination.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

FRANK S. CRUICKSHANK,
Secretary, Norfolk District Medical Society.

BOOK REVIEWS

The Climacteric (The Critical Age). By Gregorio Maranon, Professor of Medical Pathology in the Madrid General Hospital, Member of the Royal National Academy of Medicine. C. V. Mosby Co. 1929. Price $6.50.

This is an interesting book on a difficult subject. In the preface Professor Maranon states the importance of hypothesis and clinical knowledge in developing his thesis, and he certainly uses both to a large degree. It is quite true that books on the subject of the menopause are very badly needed and, therefore, this book should have a niche, but unfortunately it mixes theory and fact in such a way that it is very difficult to determine one from the other. In the vast accumulation of scientific facts in regard to internal secretions, there are certainly more specific data available than found in this book, and the continuous use of loose diagnoses in regard to changes which occur at this period of one's life seems unfortunate. The menopause is considered a complicated manifestation in which many glands play a rôle, and in which the sex glands are but a part. This is obviously an interesting and logical viewpoint, but the evidence which establishes these relationships is often inadequate and unconvincing. At the same time, the reviewer must admit that the book is exceedingly well written and has an interesting and instructive point of view.

Endocrine Disorders. By Hans Curschmann, Director of the Medical Clinic, University of Rostock. Oxford University Press. 1929. Price $4.00.

This book of Professor Curschmann summarizes in 188 pages the various clinical disturbances of the internal secretions. There are many interesting pictures of the various endocrine abnormalities. No attempt is made in this short book to discuss the recent advances made in these subjects in the laboratory. There are, therefore, a certain number of incorrect viewpoints in regard to the cause of some of these diseases. Were the physiological viewpoint more stressed, the book would be more appealing, but it certainly must be considered one of the superior books now being published in regard to these very difficult diseases. The clinical descriptions are clear and the interpretation and treatment conservative and concise. On the whole, this is a book which will strongly appeal to the average general practitioner and student who are anxious to know the present conception of these most interesting diseases, and the book is highly recommended to them. It is a striking fact that diabetes is not included in this book on endocrine disorders. It seems obvious that this disease should certainly be included.